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AUDITORY NERVE EXPERIMENTS IN ANIMALS AND THEIR RELATION TO HEARING.*

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The experiments now being carried on in the Psychological Laboratory of Princeton University by Dr. Bray and me were planned in view of the same general problem as sketched by the previous speaker. It was hoped that evidence relating to the fundamental questions of audition might be obtained by investigating the response of the auditory nerve in its relation to stimulation of the ear by sound. The object of these experiments, therefore, was an examination of the action-currents set up in the acoustic nerve of an animal during normal auditory stimulation.

The cat was selected as a suitable animal for this investigation because of its availability and convenience for operative purposes, and because of casual and experimental evidence of hearing closely approaching that of man. Some experiments performed preliminary to the present investigation showed the upper limit of hearing in the cat to lie in the general neighborhood of man's; three animals

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suitably trained were found to possess upper thresholds between 10,000 and 20,000 cycles under conditions yielding a value of 15,000 cycles for the average human ear.

The procedure of the experiment consisted of an exposure of the auditory nerve of the animal, the application of an electrode to this nerve, and the conduction of the action-currents picked up by the electrode through an amplification system to a telephone receiver. The nerve response could thus be detected by an observer who placed his ear to the receiver.

An essential feature of the investigation was the development of a suitable operative method for the exposure of the auditory nerve. Our method consists of a dorsal approach to the nerve along the inner face of the petrous bone, toward the point where the nerve emerges from the internal auditory meatus and enters the medulla oblongata. In the early work the animals were decerebrated under ether anesthesia, and the nerve approach then made, but in more recent work we have used an injected anesthetic whose effects continue throughout the course of the experiments and make decerebration unnecessary. The bony tentorium is cut away in front of the nerve-region to allow access of light, and usually a portion of the cerebellum is removed, and thus the eighth nerve and auditory region of the medulla are exposed to view. (Lantern slide demonstration of the operative field in the guinea pig.)

An electrode, usually in the form of a small wire hook, is applied to the nerve, while an indifferent electrode is embedded in some neighboring tissue, and the two wires led from the operating room to a soundproof room to connect with the amplifying apparatus. (Lantern slides of amplification system.) The output of the amplifier leads to a telephone receiver, by which the observer is able to detect the signals as sound. The soundproofing is such as to avoid any direct conduction of the stimulating sounds to apparatus or observer.

The principal results of the investigation can be stated very briefly. The experiments disclosed—we must admit, somewhat to our surprise—that the frequency of action currents as detected in the auditory nerve corresponds in a very faithful manner to the frequency of sound waves applied to the ear. A given tone sounded into the animal's ear is heard as that same tone in the receiver. The correspondence between sound-frequency and frequency of nerve impulses is such that the observer can detect no difference in pitch between a sound as heard through the preparation and that sound as transmitted directly to his ear. In other words, the ear of the

animal acts in much the same rôle as a microphone in a telephone line, translating sound-waves into action-currents in the nerve in faithful fashion. Indeed, the fidelity of transmission is such that one can understand speech with great readiness, and under good conditions even recognize the voices of persons speaking into the animal's ear. The range of tones perceptible under the conditions of amplification used is from below 120 to over 5500 cycles; responses above 5500 cycles would probably be received with increased amplification. (Several factors combine to limit the upper frequencies: poorer operation of the amplifier, reduced response of the receiver, lowered sensitivity of the ear of the observer, and possibly also a reduced response from the nerve itself.)

The results so far apply chiefly to the frequency-relations, and offer less information regarding intensity. We can say definitely that, within limits at least, louder sounds applied to the ear of the animal produce more intense responses in the nerve. Under good conditions a faint whisper in the animal's ear can be detected readily, and ordinary speech is heard as a comfortably loud signal; but as the condition of the preparation becomes impaired, through interference with the blood supply, or through drying or other injury to the nerve, the responses decrease in magnitude until they appear only to very loud sounds, and finally cease altogether.

Numerous checks have been applied in the effort to guard against any possibility of artifact. The response is definitely auditory, as it disappears on any serious impairment of the cochlear organ. It disappears on death of the animal, and temporarily ceases during restriction of the circulation to the head. The responses are blocked during the application of a steady direct current to the nerve, but return on the removal of the polarizing current. The responses can be picked up by placing the electrode on certain fairly well defined regions of the medulla and anterior portion of the brain stem; but they cease if both cochleae are destroyed, or if one cochlea is destroyed and the contralateral nerve is severed. Severing some of the fibres of the nerve under the conditions just mentioned results usually in a reduction of the intensity of the response; this experiment is complicated by the presence of vestibular as well as cochlear fibres in the nerve.

The above experiments have been repeated, with essentially similar results, on three other mammals, the guinea pig, rabbit and rat.

Some more recent work has been done on reptiles. Specimens of the common painted terrapin were used. The two branches of the eighth nerve were exposed, and an electrode applied as described

above for the cat. (Lantern slides of anatomy of the turtle.) The results were of the same general character as reported for the mammals, except that under corresponding conditions the high-frequency range was greatly restricted. Low-tone response was excellent, but the intensity began to fall off rapidly beyond 500 cycles, and signals became inaudible around 1000 cycles. Speech was transmitted with much distortion, being practically unintelligible.

Similar experiments have been attempted with frogs, but so far without results. It is planned to extend the investigation to other representative members of the several classes of vertebrates.

RELATION TO THEORY.

The relation of the results of this investigation to auditory theory is at once apparent. The representation in the nerve response of the frequency of stimulating sounds is plainly contrary to certain theories of hearing. If we can accept the evidence of these experiments, then we can no longer maintain, as does a common version of the Helmholtz type of theory, that the frequency of impulses in the auditory nerve is merely representative of intensity of stimulation, and bears no relation to stimulation frequency.

The representation of stimulation frequency in the auditory nerve, as shown in these experiments, can be explained in two ways:

1. We can assume that individual fibres of the nerve transmit frequencies as high as 5500 or more per second. This assumption leads to some form of "telephone theory," such as advocated by Rutherford. The difficulty of this view, as is well known, is its requirement in the auditory fibres of a refractory phase one-tenth or less that measured in other mammalian nerves. Many are unwilling to admit this possibility, but the question must remain open until there is more direct evidence, preferably through a measurement of refractory phase in auditory nerve fibres.

2. It is possible to formulate a theory which explains the results obtained, and at the same time avoids the refractory-phase difficulty just mentioned. This theory, which we call the *Volley Theory*, was first suggested by Dr. Harvey Fletcher to explain the facts of auditory localization in terms of phase differences. On this theory we assume that no single fibre transmits the stimulation frequency when it is great, but that large groups of fibres co-operate in establishing that frequency in the nerve as a whole. The fibres fire in volleys, in serial fashion, at a rate that is determined by the frequency and intensity of stimulation and by their several rates of recovery in relative refractory phase. And it is plain that while the fibres may be out of step with one another, they are always in synchronism with

the exciting stimulus. Hence their combined response, as transmitted by the nerve, and thus as picked up by an electrode in contact with the nerve, is faithfully respresentative of the stimulus as effective at the end organ. On this theory the refractory phase need be no different from that demonstrated for other nerves; the individual fibres need respond no faster than perhaps 1000 times per second, but yet when combined with many others in a volley might easily represent frequencies such as those revealed in the present work, and even as high as 20,000 or more per second. Intensity on this theory would be represented by the frequency at which individual fibres enter the total volley; or, in other words, the total number of impulses in the volley as distinguished from the frequency-pattern which those impulses determine. This latter feature is in harmony with Adrian's intensity-frequency principle, convincingly demonstrated in several other sensory nerves.

I cannot take time now to discuss the further implications of the Volley Theory; it is evident that we favor it as against the first theory suggested. But at the present status of our knowledge we must admit that both theories are possible, and that a final decision can be made only after further investigation of these fundamental problems of audition.

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NATURE OF STIMULATION AT THE ORGAN OF CORTI IN THE LIGHT OF MODERN PHYSICAL EXPERIMENTAL DATA. AUTHOR'S ABSTRACT.*

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The active prosecution of a program for the study of deafness has arrived at a point where a correct understanding of the mechanism of hearing may be utilized with profit. A "theory" should not be regarded as an academic description in terms of mathematical symbols of what is conceived to be a correct and final solution of the problem. It should be regarded as a necessary correlation of experimental data. The "correctness" of the theory should be judged by its utility and as long as it satisfies all demands made on it there is nothing "wrong" with it. The most that can be asked of any theory is that new experimental data, as it appears from time to time, will modify the conclusions only in quantitative detail but not in its broader qualitative aspects. The principal value of a theory is in the practical use that can be made of it, the value of it as an intellectual exercise being negligible.

The "theory" of hearing which apparently is in accord with all experimental data, whether it be anatomical, physiological or physical, is that which in its rudimentary form is known as the Helmholtz theory. Owing to the existence at present of a large quantity of precise data, particularly of a physical nature, this theory has undergone considerable advance since the time of Helmholtz.

Briefly, this theory ascribes the principal part of sound analysis to the mechanical properties of the end organ. In order to accept the essential points it is necessary to be agreed on a limited number of specific points.

1. If the basilar membrane vibrates with sufficient violence the hair cells in the superstructure of the organ of Corti are stimulated; and further if, in response to a sound, the basilar membrane vibrates more violently in one place than in another, the stimulation of the nervous tissue is greatest where the vibration is most violent.

2. The basilar membrane does vibrate in response to sound and does so differently at different frequencies. It is easily shown by

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an elementary theory of mechanics that all bodies of whatever nature, whether solids, diaphragms, membranes, rods or bodies of fluid, behave in this fashion. Theoretically, it is possible to describe a body which vibrates the same at all frequencies, but such a body is never found experimentally. This leads to the conclusion that the basilar membrane where the nerve terminals are situated is quite capable of performing an analysis of a kind of sound.

3. The vibration of the basilar membrane resulting from sound is greatest at the proximal end for high frequencies and at the distal end for low frequencies. In order to arrive at this conclusion Helmholtz depended on purely mechanical considerations, which for anyone familiar with this type of philosophy is fairly satisfactory. Histological examination of ears known to have lowered acuity in certain frequency ranges have shown this to be the case.

4. In the normal ear there is only one spot which vibrates sensibly in response to one pure frequency in the cochlea. This thesis is quite well established by measurements on masking of one pure tone by another, in which case it is found that one sound masks another more effectively when the frequency of the second is nearer the first.

5. The only sensible functioning connections between nerve cells of the spiral ganglion, either direct or indirect, through branching of the peripheral axones at the organ of Corti, are confined to near neighbors. This thesis is also established by the physical data on masking.

6. The minimum detectable change of pitch corresponds to a shift along the basilar membrane of the vibrating spot for a distance equal to the space occupied by a definite number, approximately constant, of ganglion or hair cells.

With these points taken for granted it is possible to describe the mechanism of hearing in its broader aspects and to calculate to an approximation the actual position on the basilar membrane at which different frequencies stimulate it and to calculate also the extent of the stimulating spot for each frequency.

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ANATOMY AND PATHOLOGY OF PETROUSITIS.*†

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In the consideration of the anatomy of the petrous pyramid and its apex it is perhaps advisable to classify them in terms used to designate the types of mastoid processes: namely, pneumatic, diploetic and mixed. Just as the mastoid process may vary in its structure so may the petrous pyramid and its apex vary. Zuckerkandel, a good many years ago, studied anatomically 250 temporal bones. He found that the mastoid process was pneumatic in 36.8 per cent. diploetic in 20 per cent and mixed in 43.2 per cent. Recently Belinoff and Balan[1], of Bulgaria, studied grossly the petrous pyramids of 40 consecutive cases, particular attention being given to the anatomic structure of the apex. They found the apex pneumatic in 35 per cent, diploetic in 22.5 per cent and mixed in 42.5 per cent. In comparing these two sets of figures, we find that the per cent of each type is practically the same for the mastoid as for the apex. Belinoff and Balan found that while in almost every case the anatomic structure of the apex was practically the same as its mate of the opposite side, it did not follow that in the same temporal bone the structure of the mastoid corresponded to that of the apex. On the contrary, in only 37.5 per cent of the cases was the structure of the same character in the mastoid as it was in the apex of the same temporal bone: Their structure varied in 62.5 per cent of the cases.

While it is the rule for the mastoid process to be more pneumatic than the petrous pyramid and the apex, nevertheless the reverse condition sometimes exists, namely, the mastoid process is diploetic while the paralabyrinthine cells and the cells at the apex are pneumatic. At times there may be as few as one or two cells at the apex. These anatomic variations, as will subsequently be pointed out, undoubtedly play an important role in the spread of infection from the middle ear to the apex.

While comparatively little work has been done on the gross anatomy still less has been done on the histology of the apex of the petrous pyramid. It is needless at this time to describe all the anatomic

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structures of the petrous pyramid and apex. The structures at the apex that are of great concern to the otologist are the Gasserian ganglion and the sixth nerve. As is well known, the Gasserian ganglion is situated in a depression on the anterosuperior aspect of the apex and is retained in a cleft formed by the splitting up of the dura. In this region the sixth nerve traverses Dorello's canal, which is bridged across by a ligament which runs from the apex forward to the sphenoid bone and is known as the petrosphenoidal ligament. In our gross and histologic studies of the apex we found that this ligament varies in size and shape and at times is even replaced by bone. These findings are in accordance with those of Vail[2].

Infections of the middle ear and antrum have a particular affinity for certain groups of cells. According to Neumann they are, in order of their frequency: *1.* mastoid process, *2.* fossa sigmoida *3.* tegmen tympani et antri, *4.* petrosa, including the labyrinth, *5.* posterior limb of the external semicircular canal and the carotid canal. While the predilection for the cells of the petrous pyramid is fourth on the list, nevertheless a petrousitis is far more prevalent than was previously believed. This has been demonstrated both clinically and histologically.

As regards the routes of extension from the middle ear and antrum to the apex, various paths have been described by Perkins[3], Sears[4], Chamberlain[5], Maybaum[6] and others. In the main they are along the paralabyrinthine cells (above, below and on the side of the labyrinth) along the cells accompanying the Eustachian tube, through the carotid canal, through subarcuate fossa, along the superior and inferior petrosal veins, from an extension of an extradural abscess, or from a combination of these. The infection as it spreads through the petrous pyramid may involve or avoid the labyrinth in its course. In not one of our four cases which we have studied histologically in which the Gasserian ganglion or the sixth nerve was involved was a labyrinthitis present[7]. Cases of this type, however, have been reported in which the labyrinth was involved. There is no doubt that the spread of infection through the petrous pyramid is most common by direct extension through the paralabyrinthine cells. Hence it stands to reason, other factors being equal, that the more cellular the petrous pyramid the more ideal is the situation for the spread of infection.

Although infections of the petrous pyramid and its apex may vary in intensity, not all infections in these structures include the fifth and sixth nerves. Cases have been reported in which a localized abscess was present at the apex without an involvement of the fifth

and sixth nerves. Furthermore, infections of the petrous pyramid may break through the cortex of the bone along its course and produce a localized extradural, subdural or brain abscess or a generalized meningitis, or as was previously pointed out, it may extend along the dura to involve the Gasserian ganglion or abducent nerves. Thus we have studied histologically cases in which an osteitis of the petrous pyramid was present with involvement of the fifth and sixth nerves and cases where the pathologic changes in the petrous pyramid were quite similar without involvement of these nerves. In the latter group the cases terminated in generalized meningitis or in brain abscess[8]. Also there are cases where in spite of the absence of changes in the petrous pyramid the nerves were, nevertheless, involved. Hence, the involvement of the fifth or sixth nerves is not always a criterion as to the presence or absence of an inflammatory process at the apex. It follows, therefore, that there are causes other than an osteitis for the involvement of these nerves. What the factors are which determine the various complications under the same pathological conditions remain questionable.

Just as the mastoid process, the petrous pyramid and the apex differ in their anatomic structure so do they differ in their pathologic picture. While it is the rule that the degree of infection is greater in the mastoid cells than in the paralabyrinthine cells, at times the reverse condition holds. Thus, while there may be little or no infection in the mastoid cells, the paralabyrinthine cells or the cells at the apex may be the seat of a localized collection of pus. Cases of this type have been reported by Otto Mayer[9], Karlefore Ulrich (quoted by Belinoff and Balan[1]) and others. Moreover, we have demonstrated histologically that infection may subside in the middle ear and mastoid cavity but will increase in severity as it advances through the petrous pyramid. This bears out clinically the not too infrequent occurrence of a dry resolving middle ear associated with an intracranial complication.

As regards the pathogenesis of the fifth and sixth nerve involvement, namely, the so-called Gradenigo syndrome, various theories have been expounded. Among these are localized pachymeningitis, diffuse meningitis, circumscribed suppurative leptomeningitis, serous meningitis, inflammatory edema of the tip, toxic and reflex (labyrinthine). It is our opinion that an osteitis of the petrous pyramid associated with a localized pachymeningitis at the apex, *i. e.*, a periganglionitis and ganglionitis, is a frequent cause of this syndrome. We are cognizant, however, of the other causes for this condition. Thus, it is evident that there is no single constant factor responsible

for this syndrome; the value of the term, Gradenigo syndrome, is therefore doubtful.

In conclusion, I wish to emphasize that in view of the various anatomic and pathologic changes that occur in the petrous pyramid a thorough understanding of these is absolutely essential in the application of surgical therapy.

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THE SYMPTOMATOLOGY AND DIAGNOSIS OF SUPPURATIONS OF THE PETROUS PYRAMID. AUTHOR'S ABSTRACT.*

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The symptomatology of suppurations of the petrous tip can be divided into four periods: The period of the eye pain and aural discharge; the period of low grade sepsis; the period of quiescence; and the terminal stage. As a rule, only the fourth stage appears as an entity.

THE PERIOD OF THE EYE PAIN AND AURAL DISCHARGE.

Preliminary Data: All of our cases occurred in patients whose mastoid processes showed extensive pneumatization. Before the primary operation on the mastoid process, extensive ramification of the cellular elements was always noted on radiographic examination. At operation, this observation was verified. The cells themselves, or the spaces created by their coalescence due to the disease, were found to occupy large areas of the zygoma, squama and occipital bone, and to surround the base of the petrosa completely. These findings are significant; for, by demonstrating that the process of pneumatization has extended beyond the limits of the mastoid process, they suggest the possibility that the petrous pyramid is pneumatic.

Eye Pain: This is, in the majority of instances, the first symptom to make its appearance. Its location and character are so typical that it is almost diagnostic of petrosal tip suppuration in itself where the anatomical structure already described has been found.

The pain is on the side of the lesion. It is limited to the region about the eye and is felt within the orbit itself. It is described as a deep seated ocular pain and, at the onset, is nocturnal in character.

This peculiar type of pain is highly significant of a petrosal tip suppuration. It is the result of an irritation of the ophthalmic branch of the trigeminal nerve.

In our cases eight presented this typical eye pain as the initial symptom. In one case the pain was present from the onset and was not relieved by any of the surgical procedures instituted prior to the drainage of the petrosal tip. In the remaining cases, a varying inter-

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val of time elapsed between the performance of the simple mastoidectomy and the beginning of the eye pain.

Pain in the face and teeth can occur with a suppurative lesion located anywhere in the middle ear or mastoid process. This pain will be relieved, however, as soon as the source of irritation in the middle ear or mastoid is removed. Pain felt in the region of the orbit, due to irritation of the ophthalmic branch of the fifth, must be caused by a lesion in direct proximity to this branch for it has no connection with the other sensory nerves in the petrosa.

When surgical removal of the purulent focus in the mastoid process and middle ear does not result in a cessation of the pain distributed over the areas supplied by the second and third branches of the fifth nerve, the persistence of the pain should be viewed as suspicious of a petrosal tip suppuration, when it is continuous in nature and not of the spasmodic type. Spasmodic pain is more apt to be associated with an idiopathic lesion of the Gasserian ganglion itself, like tic douloureux or neuritis. In petrosal tip suppurations we are more likely to get a constant ache than a spasmodic pain.

The Aural Discharge: It has been the experience of most otologists that a simple mastoidectomy which has established adequate postauricular drainage will cause the middle ear to cease discharging within a week or two after operation. In our cases of petrosal tip suppuration, either the middle ear continued to discharge until the lesion in the petrous tip was identified and eradicated, or else, after a period during which the ear was dry, a profuse discharge suddenly reappeared, at the same time as, or shortly before, the onset of the eye pain.

In a previous paper appearing in the Annals of Otology, Dr. Almour and I have discussed the significance of petrosal tip suppurations as the cause of a type of chronic otorrhea in patients showing a well pneumatized mastoid process. An acute middle ear suppuration which has a coalescent mastoiditis as a complication will, in most cases, undergo resolution after the mastoiditis has been eradicated. The otorrhea may persist after simple mastoidectomy on a well pneumatized mastoid process if the acute lesion in the middle ear was of the type known as the "acute necrotic otitis."

In our cases of petrosal tip suppuration, the continued otorrhea could not be accounted for in this way. Neither the otoscopic picture nor the pathological findings at operation agreed with that presented by cases of bone caries or secondary cholesteatoma.

In the performance of the radical operation on our cases, however, we were impressed with the amount of granulation tissue present in

the antrum or middle ear. In seven cases, it was situated around the tubal orifice. The tubal mucosa was markedly edematous, whereas that covering the promontory and Fallopian canal was glistening and thin. No areas of necrotic bone were present within the middle ear. In the remaining two cases, a large fistula was found above the horizontal and behind the superior semicircular canal, leading inward and forward toward the tip of the pyramid. The opening of this fistula was surrounded by granulations; and mucopus exuded from it. The middle ear was normal.

Uffenorde considers the reappearance of otorrhea in conjunction with eye pain a significant factor in the symptomatology of petrosal tip suppuration. Eagleton's cases, as well as others reported in the literature, all show this recurrence or continuance of a discharge from the tympanic cavity.

As already stated, we explain the recurrence of the otorrhea on the basis of the observations made by Lange. In most instances, the pus in the petrous tip drains into the middle ear through the channel created by the peritubal cells. In some cases, the pus drains out through the avenue of invasion, which is located in the region of the inner antral or inner epitympanic wall.

Other Symptoms and Signs Occurring During This Period: It has been our experience that the eye pain and the aural discharge constitute the salient findings which are invariably present early in the course of a petrosal tip suppuration. Other signs which are occasionally present, and which are included here as corroborative—not diagnostic—data, are transient facial weakness, vertigo, nystagmus and vomiting.

THE PERIOD OF THE LOW GRADE SEPSIS.

The temperature in our cases of petrosal tip suppuration was that of a low grade sepsis. On an average the temperature was low in the morning, between 99° and 100°. Toward the late afternoon, it would rise to 101°-102°.

Postoperative temperature following mastoidectomy may have many causes. Consequently, no significance can be attached to it unless other symptoms are also present to help identify the source of the fever. When a low grade sepsis continues after mastoidectomy, accompanied by eye pain and aural discharge, it is to be viewed as very strong corroborative evidence of a petrosal tip suppuration.

THE PERIOD OF QUIESCENCE.

In most of our cases there occurred an interval of freedom from all pain of diagnostic import. This period of quiescence varied in duration from five to 19 days. Before proceeding further it must

be repeated that the pain to which we are referring is the deep seated eye pain, associated with a low grade sepsis. As previously shown, the presence of trigeminal neuralgia alone or of pain not limited to the first branch of the trigeminal nerve in no way serves as a diagnostic symptom of petrosal tip suppuration. Therefore, the presence and subsequent disappearance of pain in the areas supplied by the second and third trigeminal branches do not create what we designate as the period of quiescence. We refer only to the presence of deep seated eye pain, in the company of low grade sepsis, and to the subsequent abatement of this pain.

From the standpoint of the patient's safety, this period is the most dangerous one, since it may lead both patient and surgeon to conclude that the lesion is clearing up. On the contrary, in the majority of our cases, this period coincided with the invasion of the endocranium. In only one instance did it signify spontaneous evacuation of the purulency in the tip through the middle ear.

When it is understood, as we have previously pointed out, that the eye pain is due to traction exerted on the ophthalmic branch because of the inflammatory swelling of the dura in the region of the petrous tip, it will also be understood that the relief of this inflammation will result in a cessation of the pain. We are familiar with the fact that the pain in an acute coalescent mastoiditis is considerably lessened when the pus in the mastoid process ruptures through the cortex and forms a subperiosteal abscess. This is exactly what occurs in cases of petrosal tip suppuration. The localized collection of pus within the apex at first causes an edema of the overlying dura with a consequent traction of the ophthalmic nerve, which is in proximity to it. As the lesion progresses, if sufficient draniage is not established through the peritubal cells or through the tract of invasion, the upper surface of the apex becomes eroded, either directly under the Gasserian ganglion or through the thin bony partition which separates it from the carotid artery.

Once a perforation has been formed, the pus makes its way subdurally in the region of the Gasserian ganglion. There results an extradural abscess, which bears the same relationship to the disappearance of pain in the lesion under discussion as does the formation of a subperiosteal abscess in an acute coalescent mastoiditis: the inflammatory tension on the dura in one instance, and on the periosteum in the other, is alleviated.

The eye pain will reappear after the petrous tip suppuration has resulted in an extradural abscess, when an increased tension is again exerted on the ophthalmic nerve: this occurs with the invasion of

the subarachnoidal space. A purulent meningitis results with a marked increase in intracranial tension which is transmitted not only to the ophthalmic nerve on the side of the lesion but to all the sensory nerves of the dura. A severe generalized headache then sets in. If the patient's life is to be saved and a meningitis averted, operative measures must be instituted prior to the stage of quiescence.

THE TERMINAL PERIOD.

The terminal period presents, in the main, the clinical picture of an acute purulent leptomeningitis.

Whether or not any of the cranial nerves in the proximity of the petrosa will be involved depends upon the point of rupture in the petrous apex. In none of our cases was the abducens nerve involved. This seems strange at first because of the deeply rooted impression that a lesion of the petrous apex produces the so-called Gradenigo syndrome. In another paper we have outlined the reasons why the Gradenigo syndrome as such is not diagnostic of a petrous tip suppuration and we have shown conversely that not all cases of petrous tip abscess produce a paralysis of the sixth nerve. Because of the fact that abducens palsy is not a constant symptom, in the sense that the retro-orbital pain, low grade sepsis and persistent otorrhea are constant, it is not to be looked upon as a necessary factor in the establishment of a diagnosis of petrous tip purulency.

When we consider that most cases presenting the so-called Gradenigo syndrome go on to complete recovery, whereas cases of petrosal tip suppuration either terminate fatally or result in a chronic otorrhea if no attempt is made to eradicate the lesion surgically, it is inconceivable that the two conditions should be considered identical. Since we have seen that suppurations of the petrosal tip occur without an involvement of the abducens, it is illogical to look for the outward manifestation of external rectus palsy as a symptom diagnostic of petrosal tip suppuration.

LABORATORY DATA.

The information which the laboratory furnishes in these cases is negligible. Only the X-ray can be used to decided advantage.

Bacteriological Flora: It is our opinion that the invading organism has no etiological relationship to the lesion in question. A streptococcus mucosus infection occurring in a patient who has no pneumatic structure in his petrosa cannot possibly produce a petrosal tip suppuration. This holds true for the other bacterial agents. In other words, the lesion in the petrosal tip depends upon the type of anatomical structure rather than upon the type of bacterial invader.

Blood: The red cells and hemoglobin are not affected to any extent

by this lesion. All our cases showed a mild secondary anemia such as is usually associated with any prolonged sepsis. The white cell count, which would be expected, under the circumstances present in these cases, to manifest a severe infection by a shifting to the left in the v. Schilling count, showed only a slight increase in staff forms and a moderate leukocytosis. Only when the endocranium was already invaded was there a sudden rise in the staff cells and a marked leukocytosis.

Spinal Fluid: In all of our cases, the spinal fluid showed normal chemical, bacteriological and cytological findings prior to the terminal stage, when a purulent meningitis appeared.

Eye Grounds: With one exception, the eye grounds in all our cases showed normal discs. In that case, a sinus thrombosis had been present, necessitating a ligation of the jugular and an obliteration of the sinus. This may have accounted for the slight blurring of the discs which was noted.

X-ray: With the exception of the eye pain, persistent otorrhea and low grade sepsis, this furnishes us with the most valuable guide that we have to the diagnosis of the lesion. We have been using the regular position for the base of the skull, which gives an undistorted picture of the entire length of the petrous pyramid.

In cases which, before the onset of the eye pain, showed an extensive pneumatization in the mastoid process, the base plate likewise demonstrated air spaces within the petrous bone and tip. By comparing the two sides, after the lesion had developed in the tip, a distinct difference could be noted. This varied from a blurring of the cells to complete destruction of the tip.

THE COURSE OF SUPPURATIONS IN THE PETROUS TIP.

A patient has an acute nasopharyngeal infection and then develops an abscess in the ear. After an interval of time, an acute coalescent mastoiditis develops. Radiographic examination reveals an extensively pneumatized mastoid process which has undergone destruction. At operation a large mastoid cavity is uncovered with diseased cells located not only in the mastoid process but in the zygoma, squamosa, peribulbar area and the region surrounding the semicircular canals.

After a period of normal convalescence, during which the temperature returns to normal, the middle ear ceases to discharge and the mastoid wound heals normally, the patient begins to complain of pain in the eye on the side of the lesion. About the same time, there occurs a sudden profuse discharge from the middle ear, but none from the mastoid wound, which by this time may have entirely healed. The patient begins to run a low grade temperature. The pain

in the eye is complained of mostly at night, and sometimes the patient has occasional dizziness and vomiting spells. A transient facial weakness may be present.

Examination at this time reveals a profuse otorrhea coming through a central perforation in the drum. There is a slight nystagmus which has no fixed direction. The fundi are normal. The blood count shows only a moderate leukocytosis with a slight increase in staff cells. Roentgenographic examination of the petrous pyramid at this time reveals distinct pathological changes in the region of the pyramidal apex. If the patient is operated upon at this time according to Dr. Almour's technique, pus will be evacuated from the petrosal tip, usually with a subsequent recovery.

If the lesion is permitted to advance, the eye pain suddenly subsides and completely disappears for a time. The patient feels well and only the fever remains. After a short time a fatal meningitis supervenes.

The variations in this picture are not numerous. The discharge from the ear, instead of clearing up and then recurring, may be continuous after the performance of the simple mastoidectomy. The period of quiescence may be totally absent or of very short duration. The case may heal spontaneously by the establishment of adequate drainage from the tip into the middle ear, in which case a chronic otorrhea will result.

It is our opinion that all of these cases should be operated upon and adequate drainage established for the pus in the petrous pyramid. If operative measures are instituted early, many of these cases will be saved the fatal meningitis which is the usual outcome of a petrosal tip suppuration.

51 West 73rd Street.

SURGICAL THERAPY FOR THE RELIEF OF SUPPURATIONS OF THE PETROUS PYRAMID.* AUTHOR'S ABSTRACT.

DR. RALPH ALMOUR, New York.

Dr. Almour first presented an historical sketch of the various surgical measures that have previously been advocated for the relief of this condition. He described the methods advocated by Streit, Mayer, Eagleton and others. He then described his mode of approach for drainage of the petrous tip which in brief can be summarized as follows:

A radical mastoidectomy is first performed and the root of the zygoma and the anterior canal wall are thinned down to an extent sufficient to bring into view the mouth of the Eustachian tube. The tensor tympani muscle and the cochleariform process are then removed. Dr. Almour here exhibited anatomical specimens which showed the relationship existing between the basal cochlear turn and the carotid canal and between the apex of the cochlea and the Eustachian tube. Directly under the roof of the pyramidal portion of the petrosa the basal cochlear turn is separated from the carotid canal for a minimum distance (14 specimens) of six mm.

With the use of a one or 1½-m.m. burr directed at an angle of approximately 20°-25° to the axis of the external auditory canal, an opening is made just in back of the Eustachian orifice, directly under the tympanic roof. The burr then passes into a pyramidal space, the sides of which are formed by the carotid canal in front, the three turns of the cochlea behind and the Eustachian tube externally. The apex of the pyramid is below and the base above. Through this space access to the petrous tip is possible for drainage of a suppurative focus within it.

Dr. Almour discussed the indications and contraindications for this procedure, stating that only injury to the carotid artery is to be feared. This can always be avoided if it is remembered that the uppermost portion of the carotid canal is below the level of the roof of the Eustachian orifice. Therefore, if the petrous tip is entered at the level described by him and in the area described, the carotid canal is avoided.

He stated that this procedure should never be used in cases wherein the abscess within the petrous tip has ruptured subdurally and

*Paper read in full before the New York Academy of Medicine, Jan. 9, 1931.

formed an extradural abscess. In such an event, the extradural abscess should be drained after the method described by Dr. Clarence Smith at this meeting.

Dr. Maybaum and Dr. Friesner have cautioned us to look out for other sources from which pus might come before we enter the petrous tip. I took that for granted in the presentation of my paper. Before the petrous tip is entered, a search is made of the peribulbar area, the area around the petrous base, etc., then when the simple cavity is converted into a radical one, all the areas which might be involved in the antrum and epitympanic space are carefully inspected and if necrosis is found these areas are probed. All these cases should be inspected very carefully to determine the area of invasion, and if that be determined, the tip should be entered if the symptoms Dr. Kopetzky has outlined are present. Dr. Maybaum calls the operation I have devised "dangerous." It is not more dangerous than the simple mastoid in the hands of a tyro; anyone who does not know the anatomy of the mastoid process should not be permitted to do a mastoid operation; and similarly, in order to perform this procedure, a clear and accurate knowledge of the anatomical structures present and to be avoided must be known. If one is familiar with these, there is no danger. The entrance to the carotid is the one danger that presents itself, but if the burr is placed at the high level described, before the superior wall of the carotid becomes membranous, one cannot injure the carotid artery. If the carotid be exposed and bathed in pus, the canal should be drained. Again, this operative entrance into the petrous tip is not advised in any case that has ruptured subdurally. If a subdural abscess is present it is a complication; and since it is such, it should be treated as any such complication; namely, by approaching the area involved and draining it directly as Dr. Smith has done.

I wish to thank Dr. Goldstein, Dr. Eagleton and the others for the very kind treatment I have received from them.

CASE OF TEMPOROSPHENOIDAL LOBE ABSCESS COMPLICATING CHRONIC OTITIS MEDIA PURULENTA.*

DR. H. M. SCHEER, New York.

M. M., Clinic No. 707,514, Manhattan Eye, Ear and Throat Hospital; admitted Nov. 18, 1930; discharged Dec. 24, 1930; on service of Dr. J. R. Page; age 13 years.

Past History: At 2½ years was ill with bronchopneumonia for six weeks. Also had measles and pertussis, and mild scarlatina. Tonsils and adenoids were removed six years ago for impaired hearing.

Present History: Impaired hearing was being treated with inflation of the Eustachian tubes, when both ears began to discharge two years ago, which continued to date. In May, 1930, patient had a severe right earache, with marked headache on the right side of the head. More discharge appeared from the ear on the next day with blood and slight temperature, following which there was gradual improvement. The child was never backward in school until about a year ago, which the teacher attributed to poor hearing. Operative interference was advised in May, 1930, to improve and interrupt the progressive impairment of hearing, to prevent intracranial complications, if none were present already, and to stop the discharge. One ear to be operated upon at each sitting. After a reasonable period after complete recovery from the present condition, surgical intervention will be advised, on the left ear.

Functional tests, done in 1929, showed:

Right		*Left*
16 ft.	Conversational voice	17 ft.
11 ft.	Whispered voice	11 ft.
2 ft.	Acoumeter	3 ft.
32 D.V.	Low tone limit	90 D.V.
0.6	High tone limit (Galton)	0.6
+18	Schwabach	+16
Air 8, Bone 25	Rinné	Air 15, Bone 30
	<———Weber———>	

*Read before the New York Academy of Medicine, Section of Otology, Feb. 13, 1931.

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Nystagmus to left,

20 seconds Rotation Nystagmus to right, 15 seconds

Tests done Oct. 6, 1930:

Right		*Left*
8 ft.	Conversational voice	10 ft.
6 in.	Whispered voice	6 in.
1 ft.	Acoumeter	1 ft.
0.8	High tone limit	0.8
32	Low tone limit	
B.C.>A.C.	Rinné	BC.>A.C
	Weber———>	

X-ray of the mastoids done and reported by Dr. F. Law on Nov. 18, 1930:

Left	*Mastoids*	*Right*
	Size	
Sclerotic	Type	Sclerotic
xxxx	Degree of involvement	xxxx
Between knee and antrum	Absorption	Around knee
	Softening	
Back	Sinus	Back

On Nov. 18, 1930, the patient was admitted to the Manhattan Eye, Ear and Throat Hospital and operated upon, a modified radical operation being done. The dural plate in the region of tegmen antri was extremely soft and necrotic, and therefore was removed. A thin layer of grayish granulations was seen on the dura, with no free pus. Dural plate was removed until normal dura was exposed around this area. Flap was cut for widening of the external meatus and the posterior wound left partly open for drainage. Three days after operation, on Nov. 21 the temperature rose to 103.2°. Patient became stuporous and had projectile vomiting. On receiving this report immediate lumbar puncture was ordered and when I arrived at the hospital, examination of the patient showed rigid neck, bilateral Kernig, and Babinski. Report of spinal fluid examination: Cloudy under pressure, increased albumin, increased globulin, sugar greatly decreased; cells, 3,900; polys., 98 per cent; two small lymphocytes, no bacteria on smear, culture negative subsequently. Patient was immediately taken to the operating room. The postauricular wound widely opened. The area of dura was dirty gray and thickened. Larger exposure of dura was made until normal dura was seen around this dirty area, except in the region of tegmen antri. The brain and dura was elevated gently with flat elevator from the floor of the middle

fossa for about three-eights of an inch and here free, foul-smelling pus was seen coming from an opening in the dura. Brain forceps inserted here went into the temporosphenoidal lobe of the brain for a distance of 4 c.m., separating the blades at intervals as this depth was approached; each separation yielded some pus from the brain. At 4 c.m. still more exuded. Perforated rubber tubing was used as a drain, which was sutured to the skin of the wound, this being left widely open.

Cerebrospinal fluid taken the next day, Nov. 22, was turbid, under less pressure, albumin increased, globulin increased, sugar decreased; cells, 1,650; polys., 95 per cent; no bacteria on smear.

Culture of pus from the brain showed mixed growth. B. coli and diphtheroids. The eye grounds were examined at daily and two-day intervals and all times were normal. Blood counts and urinalyses at various times were negative.

On day after brain operation, Nov. 22, a note on the chart shows: Temperature, 99.8°; pulse improved. Patient quite active mentally. Kernig and Babinski apparently normal. Neck rigidity about normal or markedly improved. Wound odor partly gone.

Since the day of operation the temperature varied from 98.6° to 100°. On Nov. 29 pulse went to 68, patient again vomited and became dull mentally. Removal of the drainage tube from the brain showed it to be kinked. On following into the fistulous tract in the brain, about a dram and a half of pus flowed out between the blades of the bayonet forceps. The rubber tubing was not replaced but a large siris tubing drain was inserted. Patient felt better almost immediately on release of the retention. Drainage then continued satisfactorily until the posterior wound began to granulate in so rapidly that the siris drain could not be readily placed into the fistulous tract, which was daily draining less pus. To prevent the granulations from closing over the fistulous opening, the dressing was done through a large ear speculum and the drain inserted through this into the brain and the speculum left in. Patient was discharged from the hospital on Dec. 24, 1930. When the ear speculum could no longer be used through the posterior wound because of rapid healing, the siris tubing, at this time the smallest size, was inserted into the fistulous tract into the brain through the external meatus. The tract was filling in from the bottom as the tubing was shortened each day. When the drain was removed and left out entirely, dermatization of the opening occurred, as did the entire meatus and fundus, with the exception of a small opening, from which a small amount of pus still comes. This opening is the perforation of the tympanic membrane through

which the discharge was coming before operation, and may still in time close over as a result of the modified radical mastoid operation.

Functional tests done on Feb. 7, 1931, are as follows:

Right		*Left*
2½ ft.	Conversational voice	18 ft.
0	Whispered voice	1 ft.
9 in.	Acoumeter	8 ft.
B.C.>A.C.	Rinné	BC.>A.C.
<———	Weber	
B.C. incr. 10 sec.	Schwabach	B.C. increased 10 seconds
0 (Galton)	Upper tone limit	0 (Galton)
32?	Lower tone limit	32
0	Fistula	0
25 sec. to left	Rotation	22 seconds to right

The results of a complete neurological and physical examination done on Jan. 31, 1931, by Dr. Edwin G. Zabriskie are summed up in his statement as follows: "There were no residual signs of any disturbance of either motor or sensory pathways, with the possible exception of the slightly hazy optic discs."

On date of reading this proof, May 5, 1931, the patient is still perfectly well.

522 West End Avenue.

REPORT OF CASE OF PRIMARY BULB THROMBOSIS: OPERATION THREE YEARS LATER, CIRCUMSCRIBED LABYRINTHITIS, FACIAL PALSY.*†

Dr. L. Kend, New York.

Thomas B. was referred to us by Dr. Shippman on April 4, 1927, for diagnosis and treatment. He had had measles and scarlet fever without any complications, and aside from that he had been well, up to March 20, 1927, when he developed a very severe earache, which persisted throughout the day. On the following day the family physician, Dr. DeJohn, was called in, who referred the patient to Dr. Pool at the Brooklyn Eye and Ear Hospital. Dr. Pool examined him

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that same day and stated that the earache was due to a diseased molar tooth. He was then referred to a dentist, Dr. Dubrief, who examined and X-rayed his teeth and finding no pathology or infection, advised against removing any tooth. On the following day the mother returned and insisted that the tooth be extracted, since there was no abatement of the severity of the pain. For the succeeding five days the patient was under the care of the family physician, who administered tablets for the pain. On Monday, March 28, the mother brought the patient back to the Brooklyn Eye and Ear Hospital, at which time he was referred for an X-ray examination and told to return on Wednesday. On Wednesday a distinct swelling appeared on the neck behind the ear. That afternoon, at the hospital, the mother was advised to consult a general surgeon, since it was a case of glandular disease. (X-ray report: Slight sclerosis, left; otherwise, negative.)

Dr. Hallerhan was consulted the following day and referred the patient to Dr. Ponemon for another X-ray examination of the mastoids, who reported: "Left mastoid cloudy throughout but cell walls intact." On Saturday the patient was sent to Dr. Shipman, who sent him to us on Monday, April 4.

Examination at this time showed a boy, age 9 years, who looked acutely ill and apparently suffering a great deal of pain. The ear fundus was essentially negative, there being a little redness of Shrapnell's membrane. There was a definite fluctuant swelling below and behind the tip of the left mastoid. The case was diagnosed as acute masked mastoiditis with a Bezold perforation, and the child was admitted for immediate operation.

Operation: The removal of the mastoid cortex revealed an apparently normal diploetic type of mastoid process, and for the moment I was under the impression that I had opened a normal mastoid. The original incision was enlarged to include the abscess in the neck, from which pus flowed freely. Careful probing disclosed a large necrotic fistula in the digastric groove extending into the mastoid process. The operation was continued from above and no pathology was encountered until the sinus plate was reached. This was black, and when removed there was a completely collapsed sinus wall, free pus and a little blood. The jugular was resected and the sinus opened. A good sized clot removed from the trochlea end resulted in free bleeding. No bleeding, however, could be obtained from the bulbar end, although a great deal of organized clot was removed. Since the patient's pulse was growing thin and rapid, it was decided to stop at this point. The boy was removed to bed, and 300 c.c. of normal

saline given hypodermically. He made an uneventful recovery. A smear of pus showed long-chain streptococci.

The patient was seen again on May 12, 1930, with the left ear discharging and the region of the mastoid red, swollen and tender, with definite fluctuation. His face was drawn over to the right, and he was unable to perform any muscular movements with the left side of the face. The following history was obtained: He had been well until early January, 1929, when he had a vomiting attack, which persisted for three days. A diagnosis of acute appendicitis was made but operation was delayed until the fourth day, when the vomiting ceased and the left ear began to discharge. The diagnosis of acute appendicitis was changed to "gastric poisoning," and after two days the discharge from the ear stopped and the patient was apparently well. During the winter of 1930 the boy complained of occasional pains in the old scar in the region of the mastoid. On May 5, 1930, the left ear again began to discharge. On May 10 the mother noticed the weakness in the left side of his face, and on May 12 the mastoid region became as described when the boy presented himself for examination. *Diagnosis:* Acute secondary mastoiditis and facial paralysis, left.

Operation: Incision along old scar; pus under pressure poured out; granulations removed, disclosing a fistula in the lateral semicircular canal and another into the Fallopian canal just below the lateral semicircular canal. Probing the latter resulted in a twitch of the facial muscles on that side. The facial nerve was uncovered in its perpendicular course through the mastoid, and a radical mastoid was performed. Again the patient made an uneventful recovery, though it was four months before he recovered complete facial control. There still remains a spontaneous nystagmus on looking to the right.

Postoperative Diagnosis: Acute secondary mastoiditis, left; facial palsy, and circumscribed labyrinthitis, left.

AURAL CHOLESTEATOMA WITH CASE REPORT.

Dr. Edward F. Ziegelman, San Francisco.

Cholesteatoma may be defined as an encysted tumor containing cholesterin. This broad definition applies to the formation of such a tumor in any portion of the body. When aural cholesteoma is mentioned we usually think of that form of new growth which is apt to originate in the middle ear. It may extend in any direction; the mastoid process is the usual site of the secondary invasion. Ballenger in his recent textbook states that "cholesteoma of the middle ear is characterized by the formation of masses of epidermoid cells arranged in concentric layers between which are found cholesterin crystals."

This condition in the ear may be the result of either a primary or a secondary new growth. The primary type is exceptionally rare. A few cases have been reported in medical literature. In questioning workers in large otological clinics or with large private practices, I have never found one who has seen a case. No doubt the primary type does occur but it must be considered a pathological curiosity.

A far different story may be told regarding the secondary type. A safe rule to follow is "every case of chronic purulent otitis media is a cholesteamatous possibility." The belief prevails that this pathological menace materializes in a very small percentage of chronic discharging ears. I believe if we could study the pathology in the living by the radical mastoid route in a series of chronic discharging ears we should be astounded at the microscopic findings. Certainly, cholesteatoma is more prevalent than we are led to believe.

Preventive medicine has been the watchword of the past half century. Of course, this should include preventive surgery. At times words are interpreted too literally. The prevention of surgical conditions or at least prevention of their sequelae is too often neglected. This applies to the treatment of chronic purulent otitis media. In justice to our specialty, the lack of co-operation on the part of the public is responsible in great measure for this condition.

American oto-rhino-laryngologists may well be proud of the pioneer work and of the continued insistence upon prevention of medical and surgical aftermaths of ignoring infections about the head and neck. The sound, logical address given by Dr. John F. Barnhill, President-elect of the Academy of Ophthalmology and Otolaryngol-

ogy, at the Atlantic City meeting in October, 1929, should be studied by all holding a medical degree. Unfortunately, addresses of this kind do not come to the notice of general practitioners, who, after all, have the greatest possible opportunity to spread such teaching.

With the exception of those rare conditions known as specific granulomata, every chronic purulent otitis media has been at some time acute (see Fig. 1). Consequently, every secondary cholesteatoma is a sequelae of an acute otitis media. It follows in so high a percentage of cases that we are justified in doing everything possible to prevent acute infection of the ear passing into the so-called chronic stage, even to the radical procedure of advising an earlier simple mastoidectomy. I know this statement will cause a furor among some of my colleagues. Analyze the unsatisfactory results in our cases of chronic purulent otitis as well as the great danger to life in its somewhat frequent secondary pathology, cholesteatoma, and the old saying, "penny wise rather than pound foolish," is applicable.

As to the etiology of this condition, many hypotheses have been advanced, each having its advocates. The prevalent theories are:

1. That it is congenital, similar to the lining of the dermoids.

2. Haberman believes that it is a secondary invasion of the skin through a perforation in the drum, an opinion shared by Prof. Georges Portmann, of Bordeaux.

3. Uffenorde believes it to be a metaplasia.

4. Manasse believes that it is a tumor implantation.

5. Fisher, of Alexander's Clinic, believes it to be analogous to skin cancer (see Figs. 2 and 3).

Regardless of the theory favored, most otologists agree on the following facts:

1. That it is relatively common.

2. That it is dangerous.

3. That intracranial complications are prevalent.

4. That it has a great tendency to grow (see Fig. 3).

5. That it causes bone destruction and atrophy.

6. That a suppurative process is always around the cholesteatoma (see Figs. 2 and 3).

The diagnosis resolves itself into a careful history and examination of the ears with an examination of the mastoid by a good comparative X-ray study. When possible there should be a pathological study of a small piece of tissue from the ear. I say when possible, for there are cases in which any instrumentation in the ear is dangerous. The presence of a fistula is important, though of course its absence has no diagnostic value. When intracranial or labyrinthine

symptoms exist indication for immediate intervention is evident. These last symptoms are of some value in leading one to suspect a cholesteatoma providing the disease of the ear is of long standing.

I have found the secretions and tissue debris, obtained from the middle ear with a cotton applicator and floated on a microscope slide where a few drops of water have been placed, with the demonstration of shining crystals of cholesterin, simple, practical and satisfactory. Chloroform solution on the slide, with the change of color to greenish, has not proved as positive for me as the probably less scientific method of the crystals on an aqueous mount. In one case, referred pain in the lateral pharyngeal wall was very interesting and led me to suspect the possibility of a middle ear cholesteatoma, which X-ray and water mount confirmed.

Fig. 1. Photograph of drawing which shows the prevalent site and type of perforations from which a cholesteatoma develops. Note the location of the perforations, that is either marginal or in membrana flacida.

The treatment of this disease is a complete evacuation of the mastoid cavity by the radical procedure following the diseased condition to its utmost ramifications. In this I have been greatly aided by the use of the binocular eye loupe. It is well known and demonstrated by microscopic study that cholesteatoma is apt to invade the Haversian system, so, at best, we can never be sure that the diseased tissue has been completely removed. When thorough evacuation has been attempted the liability of recurrence is much less than the chances of complete recovery.

I have found the use of chlorin solutions to be most satisfactory in postoperative care. Hypochlorite solution has taken the place of Carrel-Dakin, due to the difficulty in obtaining a satisfactorily stable product. The posterior wound is closed after the plastic flaps are

made unless meninges, sinus or canals are exposed. A large rubber tube is inserted through the external meatus into the cavity. Around this is gently packed vaselin gauze. Chlorin solution, properly diluted, is instilled through the tubing three times a day. The bandages are not removed as the tube is allowed to be of some length and is plugged with a sterile cork. It is covered at its distal end by one or two turns of the bandage. Whether this chlorin preparation has a destructive effect upon the cholesteatomatous tissue I am not prepared to say at present. I can state definitely that postoperative wounds of the infected type do much better, in our special field and in that of general surgery, when chlorin solutions are used. The virtue of this I hope to explain at some future date.

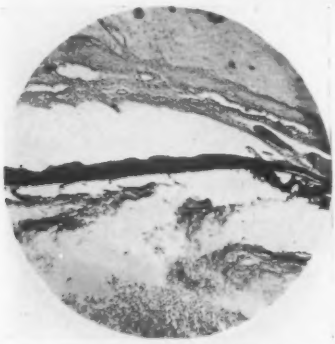

Fig. 2. Photomicrograph of microscopic section of tissue removed from case reported. Note the central area with epidermoid cells, in which can be seen cholesterin crystals. Throughout section can be seen numerous pus cells and cellular infiltration.

Case Report: F. F., male, age 19 years, white. *Family History:* Negative. *Past History:* Measles at age of 6 years. At that time left ear ruptured spontaneously. Since then has had a discharging left ear at intermittent periods. *Present History:* While playing center on his high school team, noticed he became dizzy at times when stooping over. Had to relinquish his position on the team. Consulted me for dizziness.

Status Praesens: A well built young male adult, somewhat pasty in appearance, general physical examination negative. Urine negative. Blood count as follows: R. B. C.; 3,400,000; hemoglobin, 80 per cent; W. B. C., 11,500; polys., 69 per cent.

Nose: Right nares: Septum deviated to right. space fair, turbinates engorged, middle turbinate touching septum, no anterior dis-

charge. Left nares: Septum showed marked deviation in lower portion to left, space poor, turbinates normal, though inferior touched septum, due to marked deviation in lower half, slight anterior purulent discharge.

Nasopharynx: Right choana normal, left showed some purulent discharge in same, marked hyperplasia of left posterior inferior turbinate tip.

Buccal Cavity: A moderate amount of dental sepsis.

Throat: Pharyngeal tonsils embedded and septic, pillars markedly congested.

Larynx: Vallecula slightly congested, pyriform fossa contained on both sides a moderate amount of mucopurulent secretion. The

Fig. 3. Photomicrograph of microscopic section of labyrinth of petrous pyramid removed at autopsy. A nonoperative case. Location of section cannot be positively determined, but probably through a portion of the vestibule. In upper half notice the bony destruction, in lower half of photomicrograph notice the mass of cholesterin crystals near center, with other smaller areas. Also a great amount of cellular infiltration can be seen. Many pus cells were present.

epiglottis slightly congested. The cords and arytenoids somewhat congested, though both freely movable.

Ears: Right external auditory canal normal, membrana tympani retracted, slightly fibrous, poor light reflex. Postauricular region apparently normal, as well as mastoid process. Left external auditory canal had a small amount of purulent secretion on floor of canal. The membrane was retracted in the lower portion. It was somewhat reddened and no light reflex was present. In the posterior half of Shrapnell's membrane a small perforation was present, no secretion was visible. On using a Siegle, a small amount of purulent material was obtained from the perforation. The odor of the dis-

charge was foul. Aqueous slide of secretion showed some shiny particles floating on the surface of the water.

Functional Tests: Hearing, right ear: Watch, 18-36; Weber to left, Rinné, +; C1, diminished 20″; C4, diminished 12″. Left ear: Watch on contact; Weber to left; Rinné, negative; C1, absent; C4, diminished 10″. Vestibular: Spontaneous, no nystagmus, slight ataxia, no Romberg, no adiadokocinesis, no asnergy, normal gait. Pastpointing apparently normal with either hand: Induced: Turning to right, head ant. 30°, 10 x 20 sec. Hor. nystag. to left, duration 24 sec., good amplitude; first, second and third degree. Turning to left in same position and at same rate, nystag. hor., duration 20 sec., good amplitude in all three degrees. Pastpointing, after turning, 10 x 10 sec. R. hand to R., 4, 4, 3, 1, touch. L. hand to L., 3, 3, 1, touch. Caloric not done.

Fistula Test: Questionable. At times I thought I obtained an atypical hor. nystagmus to the opposite side, though it was recorded as negative.

X-ray of mastoids stereo by Dr. Howard P. Mawdsley, of "The Mills Memorial Hospital, San Mateo," showed a definite breaking down of the cells of the left mastoid.

Clinical Diagnosis: Deviated septum, chronic sinusitis, chronic tonsillitis, chronic otitis media purulenta left, with chronic mastoiditis and cholesteatoma. The last opinion was based upon the history, type of discharge and the water slide amount.

Advice: Left radical mastoidectomy, later other nose and throat pathology to be remedied.

Treatment: A classical left radical mastoidectomy was performed, a mushy granulating tissue removed from the mastoid cavity with evacuation of the cells. The dura exposed over the tegmen in following the abnormal deposits, also in the area of Trautman's triangle. After completion of evacuation, T-shaped postauricular flap made. Posterior wound left open. Progress good. Left hospital in three weeks, eight weeks good epithelization. Has been seen bimonthly during the past two years, cavity dry except that small amount of epithelial debris is removed about every four months. General condition good, no ear symptoms.

Comments: This was a case of chronic mastoiditis with cholesteatoma. It might very easily have been called chronic purulent otitis or chronic mastoiditis. Even though the diagnosis was provisional, the pathology in the living demonstrated such a condition to be present.

Space will not permit me to give detailed case histories. Suffice it to say that during the past three years I have had six cases, two proved at operation, the other four with provisional diagnosis of cholesteatoma. All were advised to have the radical operation. These patients had had treatment for years, varying from red medicine to zinc ionization. Many such patients consult an otologist solely to have wax removed.

CONCLUSIONS.

1. Cholesteatomatous growths in the middle ear and mastoid cavity are fairly common.

2. Many chronic purulent otitis medias are cholesteatomas, all are potentially.

3. Material placed upon a glass slide with water is of some value in determining the diagnosis.

4. Extensive radical evacuation of the diseased substance is important in obtaining a satisfactory result.

5. The use of the binocular loupe is advantageous in determining the extent of the lesion.

6. A more radical attitude in advising simple mastoidectomy to overcome the possibility of development of a chronic purulent otitis media.

7. A more careful examination and history of the ear, nose and throat patients who consult us.

450 Sutter Street.

TWO CASES WITH SEVERE LABYRINTH SYMPTOMS OCCURRING FOLLOWING O. M. C. C.*

Dr. John McCoy, New York.

Case 1: Dr. J. P. V. age 45 years, came to me in March, 1930, complaining that he had an attack of streptococcus sore throat and otitis media catarrhalis in both ears in the preceding December. At that time he was treated by Dr. A. L. C. He came to me in March complaining that his hearing was deficient, especially so in his left ear, and that he was suffering with attacks of dizziness, nausea and tinnitus. Dr. C. removed his tonsils some eight or nine years before.

His general habits were very good, as he never had used tobacco and never had used alcohol. He was married and had four healthy children. He gets headaches off and on since the beginning of this attack. His hearing began to be impaired, he thinks, in March, 1929, especially in the left ear.

Examination showed that his eardrums were thickened and retracted, that his Eustachian tubes were stenosed, especially the left, that his nose was in good condition. His blood pressure was low, 107; the urinalysis was negative, his Wassermann was negative, and his audiogram tests showed a loss of 24 per cent in the right ear and 47 per cent in the left ear. I sent him to have his teeth examined and his X-rays showed that they were all O. K. except one apical abscess, which he had removed. The depressing part of his symptom complex was the vertigo and nystagmus, which came on him at varying times, never going more than a week without an attack.

The tests of the vestibular apparatus showed that he had no spontaneous nystagmus. On turning to the right he had fine nystagmus, of a duration lasting 10 seconds, on turning to the left he had a fine nystagmus, lasting 12 seconds. On caloric test he had a nystagmus in the right ear, after douching, for 40 seconds, fair in amplitude. He touched with the right hand and past-pointed four inches with the left hand. With the head back he had a good vestibular nystagmus, past-pointed four inches to the right with the right hand and four inches to the right with the left hand.

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On cold douching the left ear he had a slight nystagmus after two minutes and past-pointed two inches to the right with the right hand and four inches to the left with the left hand.

With the head back he had a fair nystagmus, no past-pointing with the right hand and no past-pointing with the left hand, showing a labyrinthitis involving the horizontal canals.

He is that way to this day and has had to give up his practice, as he never knows when an attack is coming on him.

Case 2: R. R. M., age 55 years, first seen on Oct. 15, 1930, complaining of dizziness, nausea and tinnitus. This began approximately five or five-and-a-half years ago. One type, when sitting still would become dizzy and sick, the other type began about 1924. When sitting above and looking down at anything he would become very dizzy. Attacks come on two or three times a week. He has had his teeth completely attended to, had a radical antrum operation in January, 1930. He becomes dizzy when reading the paper.

His eardrums are thickened and retracted and the Eustachian tubes are stenosed. He has had his tonsils removed. His blood pressure is 120 to 135, his blood Wassermann is negative. His audiogram test shows, on his right ear 44.5 per cent loss, and on his left ear 20 per cent loss.

Vestibular Apparatus: No spontaneous nystagmus. On turning to the right amplitude is good and duration is 23 seconds. On turning to the left his amplitude is good, duration is 23 seconds. The caloric test with cold water shows no reaction after four minutes in the right ear. The douch in the left ear shows fine nystagmus after one minute and 30 seconds. He touched with the right hand and touched with the left hand. With the head back he has a good nystagmus and points two inches to the right with the right hand and four inches to the left with the left hand. This case illustrates labyrinthitis involving the vertical canals.

730 Fifth Avenue.

A CASE OF JUGULAR THROMBOPHLEBITIS EXTENDING INTO THE INNOMINATE.*

DR. WRIGHT MACMILLAN, Passaic, N. J.

The case to be described is one of jugular vein thrombophlebitis originating in mastoid suppuration and extending into the innominate vein. The apparent rarity of this condition diagnosed in the living subject is my reason for hoping that this description might be of interest to the present gathering. A search of the recent literature in English, German and French reveals only one case extending so far down.

This was reported by Dean W. Myers, of Ann Arbor, Mich., in the *Journal of Ophthalmology, Otology and Laryngology* and read before the annual meeting of the Society of Ophthalmology, Otology and Laryngology at Cleveland in June, 1920. In his case a firm clot extended below the level of the incision, which was to the sternum, and probing into the thorax through the clot brought forth no bleeding. The innominate vein was not identified. The patient recovered.

My patient, a 21-year-old man, was admitted to St. Mary's Hospital, Passaic, N. J., and seen by me for the first time, Sept. 21, 1929. He was poorly nourished, had a very toxic appearance, and complained of violent pain in the left mastoid region, chills, fever and weakness.

He gave a history of obstructed nasal breathing, for which, under local anesthesia, tonsillectomy was done five years previously. The tonsils were enlarged but not subject to acute inflammation, and were removed probably with the idea of lessening nasopharyngeal congestion. A few days after the operation he developed acute purulent otitis media on the left side, which ruptured spontaneously through the drum. Since that time the ear has been draining, with occasional short remissions. In 1926 the patient was confined to the hospital for one month, with a fever, the identity of which he does not know, but he suffered no pain during its course and his attention was not directed to his ear.

For three or four months before I saw him, the left ear had been deaf. Four days before admission the ear stopped running and

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became very painful. He was seen by an ear specialist and the drum presumably incised, but the pain continued with increased severity. There was little discharge and he was having chills, fever and sweats two or three times a day.

Physical examination on admission to the hospital showed a temperature of 105.6°, pulse of 90, and respirations, 22. There was very little edema or redness, but marked tenderness over the left mastoid. Thick, greenish-yellow, foul-smelling pus filled the ear canal. When this was removed the posterior canal wall was seen to be violently red and edematous, permitting only a minute section of drum to be seen. The eyes showed nothing abnormal. The nasal septum was deviated, partially obstructing the right side. The tonsils were out. There was some enlargement of the anterior cervical lymph glands, more marked on the left; there was no tenderness or rigidity of the neck; the heart, lungs, abdomen and extremities showed nothing abnormal. The reflexes were normal.

The urine analysis was normal except for a specific gravity of 1.040 and a deep amber color. The white blood count was 22,000, with 85 per cent polymorphonuclears.

At operation the mastoid cortex was found to be very dense but not excessively thick. The cellular bone was necrotic and much viscid, grayish, foul-smelling pus was released from pressure. On scooping out the fragmented cellular bone and removing some resistant portions of inner table over the sinus, an area of granulation was exposed, covering 2 c.m. of the sinus wall. The lateral and posterior semi-circular canal walls were necessarily exposed in clearing out the necrotic debris, as was also the cerebellar dura anterior to the lower portion of the exposed sinus wall.

The sinus was uncovered to healthy dura above and below and carefully palpated to detect resistance caused by a clot. The wall seemed uniformly soft. The wound was loosely packed with iodoform gauze and partially closed.

The day after the operation the patient's temperature fell to 101°; the second day it rose to 103°, but promptly fell and continued around 100.6° for four days. On the sixth day there was a rise to 103°, which continued to the tenth day. There began at this time sharp fluctuations between 101° and 104°.

Marked rigidity of the neck suggested that an intracranial complication be considered along with sinus thrombosis, although tenderness anterior to the left sternomastoid muscle pointed to thrombosis as the probable diagnosis.

Spinal puncture showed the fluid under pressure of 18 m.m. of mercury. Firm pressure over the middle of the anterior border of the sternomastoid muscle on the right caused the pressure to rise to 26 m.m. Similar pressure on the left side showed a rise to 20 m.m. This suggested obstruction in the left lateral sinus or jugular vein.

The spinal fluid globulin and cell count were normal. The white blood count was 23,000, with 88 per cent polymorphonuclears. The urine showed, as an abnormality, a small amount of albumin, and blood cultures taken on the seventh day were still negative on the tenth. Cultures of the perisinus pus taken at the first operation showed staphylococcus aureus, B. pyocyaneous, and pneumococcus.

A diagnosis of left jugular thrombosis was made and the operation was done on the tenth day. The mastoid wound was covered with wet bichlorid dressings and the jugular vein exposed and ligated above the common facial vein and at the lower angle of the wound. The common facial vein was separately ligated and cut as far away as possible and was seen to contain an occlusive thrombus. The lower end of the sectioned jugular was also seen to be filled with clot. The incision was extended to the episternal notch and the vein dissected out, sparing the communicans cervicis nerve and the anterior belly of the omohyoid muscle. The clot apparently involved the entire wall of the vein to the thyroid branches which were ligated and cut and found to be thrombosed. Two centimeters below the middle thyroid the jugular thrombus appeared to become mural, forming a wide, grayish streak of thrombophlebitis on the anterolateral aspect of the wall. This was traced down behind the medial end of the clavicle, taking care to avoid the posterolateral side of the vein and the thoracic duct which was not exposed. Finally, in trying to trace and remove all the diseased vein, the innominate trunk was seen going off at an angle to the right. The grayish streak marking the location of the thrombophlebitis extended onto the surface of the innominate. The collapsed portion of the vein ended about 1.5 c.m. from the innominate, apparently at a valve. It was ligated 1 c.m. above this and the dissected section removed. The cut end showed a clot 0.3 x 0.2 c.m. adherent to the discolored area on the wall of the vein.

A slender rubber dam drain was placed along the vein bed and the wound closed with clips, the drain protruding from the lower angle. A protecting wet bichlorid compress was applied and the sinus operation begun.

The original mastoid incision was easily ripped open and a posterior cut made at right angles. The sinus had abscessed and rup-

tured. The thrombosed emissary was exposed and cleaned out. The little remaining bony covering of the sinus was removed below to the limit of the mastoid cavity. The upper segment was uncovered to healthy dura only 1 c.m. above the knee. The lower limit of healthy sinus wall at this point was incised and the freely bleeding vessel was collapsed with a roll of iodoform gauze packing and the clot below removed. The mastoid cavity and jugular bulb were irrigated until clean and the necrotic sinus wall clipped away. The wound was packed with iodoform gauze, the new posterior incision was closed and the remainder left open.

Within the next 24 hours the temperature steadily dropped to 100.6°. For three days there was a daily fluctuation between 100° and 102.2°. On the tenth day the temperature rose to 103.8° but dropped to 101° the same day. On the twelfth day the temperature became normal and did not rise again. He was discharged from the hospital, Oct. 20, one month after admission.

The patient now is working steadily, looks well and weighs more than he has in six years. There is a slight discharge from the ear but with this ear he can hear conversational voice at a distance of 15 feet.

23 Passaic Ave.

STRICTURES OF THE ESOPHAGUS. DIAGNOSIS AND TREATMENT.*†

DR. GABRIEL TUCKER, Philadelphia.

By far the most frequent cause of stricture of the esophagus is caustic alkali burns. Guisez reported 185 cases of cicatricial stenosis in 2,500 cases of esophageal disease diagnosed by esophagoscopy. One hundred sixty-nine of these 185 cases were due to caustic burns. Zimanyi reports from the Budapest and Berlin hospitals, 10 per cent of patients in the nose and throat departments with caustic burns of the esophagus from lye, which the people of Hungary and Germany use in home soap-making. Other causes of cicatricial stenosis of the esophagus that may be mentioned are burns from acids, bichlorid of mercury and ammonia. Systemic diseases such as typhoid fever, diphtheria, lues and tuberculosis, scarlet fever and various pyogenic conditions produce ulcerations, followed by cicatrices of the esophagus. In a previous paper I have reported three cases of cicatricial stenosis of the esophagus in young adults following typhoid fever. Da Costa states that 15 per cent of the cases of scarlet fever may result in cicatricial stenosis. Peptic ulcer of the lower end of the esophagus may be a cause (see Fig. 1). Congenital stenosis would seem to be more frequent than the statistics indicate. It is frequently undiscovered until a foreign body lodges, when the Roentgen ray and esophagoscopic studies reveal the true nature of the esophageal lesion. A cicatricial lesion may develop at the site of congenital narrowing (see Fig. 2).

Age of Patients: The greatest number of lye burns occur in children. In adults stenosis is usually due to poison taken accidentally or with suicidal intent.

Location of Structure: Strictures that are due to caustic burns are found most frequently at the levels of the anatomic or physiologic narrowings of the esophagus. Jackson states that they occur most frequently at the level of the crossing of the left bronchus, next in the region of the cricopharyngeus, and next at the level of the hiatus.

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*From the University of Pennsylvania Bronchoscopic Clinics at the Hospital of the University of Pennsylvania and Graduate Hospital, Philadelphia.

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Mosher mentions the frequency of cicatricial stricture at the beginning of the liver tunnel.

Esophagoscopic Appearance: On direct examination of the strictured area the esophagus is usually found to be dilated above the stricture level with a pouch deeper on one side than the other. The entire strictured area is paler than the surrounding mucosa. The scar itself, being pale or white, may be annular, entirely surrounding the lumen; more frequently, however, it is eccentric, involving only a part of the circumference of the wall. The scar may be a hard

Fig. 1. Cicatricial stricture complicating peptic ulcer of the lower end of the esophagus. Treatment by esophagoscopic dilatation with local medication to the ulcer cured the patient.

ridge level with the mucosa, or even indented. The dilated portion of the esophagus may show inflamed mucosa, ulceration and frequently granulation, especially if the case presents itself soon after the injury. In multiple stricture there is dilatation between the strictures with retention of food and secretions.

Prognosis: Where the stricture is untreated the mortality is very high, and if left to itself is eventually fatal. The stenosis of caustic burns is more severe, hence more serious. In a large number of

cases reported by von Acker before the days of esophagoscopy, a mortality of 40 to 50 per cent was given. It may be said that the sooner the case comes under observation and treatment the better the prognosis. Early gastrostomy, getting the string through the esophagus before it closes, plus retrograde dilatation with the author's bougie, will cure practically all these cases, unless some renal, gastrointestinal or other systemic effect of the accident arises.

Symptoms: The immediate symptoms in lye cases are due to the severe inflammatory reaction, swelling and ulceration, which produce

Fig. 2. Cicatricial stricture of the esophagus following a congenital stenosis. Patient was treated by peroral esophagoscopic dilatation.

inability to swallow. The acute symptoms usually subside the first week or fortnight. Swallowing may become practically normal. Then gradually dysphagia develops, its rapidity depending on the extent of the injury to the esophagus. First difficulty with solids, later with liquids, and finally inability to swallow even saliva. Pain is not usually a symptom. Loss of weight is progressive, due to insufficient food. Dehydration results from inability to take water.

Diagnosis: First, history of burn or trauma is to be considered. The possibility of cancer, aneurysm, compression, stenosis, foreign

body, diverticulum and cardiospasm should be excluded before a diagnosis of cicatricial stenosis can be made. The employment of the bougie blindly as a diagnostic means in dysphagia or obstruction is not justifiable at the present day. After mirror examination of the pharynx, the next step is Roentgen ray of the neck, chest and swallowing function. This should include both fluoroscopy and films with opaque mixture. The methods of Roentgenologic study are familiar

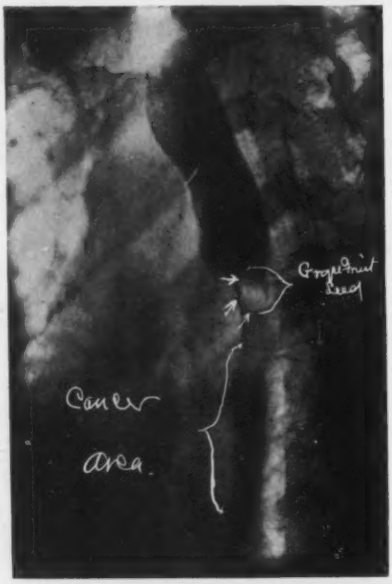

Fig. 3. Case of esophageal obstruction diagnosed "stricture." X-ray examination showed obstruction in the lower thoracic esophagus with an oval shadow above in the opaque mixture. Esophagoscopic examination revealed a grapefruit seed lodged above a cancerous infiltration of the esophageal wall. Esophagoscopic removal of tissue showed squamous cell epithelioma. The final steps in diagnosis of every case of esophageal obstruction should be Roentgen ray study and direct inspection with the esophagoscope. Temporary lodgment of food in an organic lesion of the esophagus is frequently diagnosed as "spasm." Nearly all cases of cancer have been treated as spasm.

to all Roentgenologists and need not be given in detail here. In the clinics at the University of Pennsylvania Hospital, Dr. Pancoast and Dr. Pendergrass, and the Graduate Hospital, Dr. Pfahler and Dr. Cohen, are consulted in every case, and no esophagoscopy is done until their opinion is received. After careful Roentgen ray study, esophagoscopy should be done in every case. The only exception to

this is the extremely dehydrated patient, where transfusion, hypodermocylsis and all modern means for combatting acidosis and alkalosis should be used to improve the patient's general condition. Even gastrostomy may be required in cases of this type, prior to esophagoscopy. In other words, the patient should be treated first and the stricture afterwards. The first step, however, in treating the esophagus after the patient has revived, is a thorough esophagoscopic examination. Where there is severe inflammatory reaction, lavage of the esophagus, with the administration of bismuth subnitrate, with the addition of a little calomel dry on the tongue, is very effective in clearing up the inflammatory condition.

Fig. 4. Recovering the swallowed string with a pillar retractor. The bobbin string is fastened on the neck, the string carried over the ear through the anterior nares and pharynx and through the esophagus. Adhesive plaster is placed over the string to prevent the child pulling it out of the nose.

Fig. 5. The Gabriel Tucker retrograde esophageal bougie. It is made of the best grade rubber vulcanized on a braided silk string. The braided silk allows the rubber to stretch as it is pulled into the stricture, making its diameter slightly smaller. Thirteen sizes are made, from 10 to 34 French. The length is 35 c.m.

Gastrostomy by the Mueller, Kader or Shallow method is done by the surgeon, low enough in the left rectus to permit easy access to the hiatus esophagus.

Methods of Treatment: The intolerance of the esophagus to surgical manipulation is generally recognized. The esophagus contains the infectious secretions from the mouth and in addition shock out of all proportion to the severity of the lesion may be produced by operation. This is shown in the reaction in acute traumatic esophagitis. The importance of putting the esophagus at rest where there

is marked narrowing of the canal and inability to swallow sufficient nourishment cannot be too strongly emphasized. Jackson and Mosher both advocate early gastrostomy.

Methods of treatment may be considered under: *1.* peroral methods; *2.* retrograde methods, and *3.* external operative methods. External esophagotomy, surgical reconstruction of the esophagus and internal esophagotomy have been reported as successful, but each success has been priced by high mortality. These procedures should

Fig. 6. The Gabriel Tucker bougie in the strictured esophagus. All stricture levels are dilated each time a bougie is pulled into the esophagus.

be reserved for the cases in which the other methods of treatment have failed, and it should be remembered also that a patient may live a long and healthy life under proper gastrostomy feeding.

Peroral Methods—"Blind Bougienage": The value of the method of treatment of cicatricial stricture of the esophagus of pushing down a bougie blindly, is best expressed by Trousseau, who said,

Fig. 7. A and B. Groups of children with lye strictures of the esophagus from the Hospital of the University of Pennsylvania Bronchoscopic Clinic. All the children were cured by retrograde continuous string bouginage with the author's bougie, with the exception of the boy marked X. This child had a complete atresia of the esophagus that it was thought not advisable to perforate.

"Sooner or later all cases of stricture of the esophagus die by the bougie." This observation was made, of course before the days of esophagoscopy and sight-guided esophagoscopic bouginage. On the string-guided or wire-guided peroral methods those of Sippy, Mixter and Plummer may be mentioned. Dr. Amstead Crump of New York had a very ingenious wire-guided method. These methods still have their advocates, and while they are safer than blind bouginage, they

Fig. 8. Stricture of the lower half of the thoracic esophagus. In this case attempts at peroral dilatation failed, the patient was unable to swallow a string, and the string was passed by retrograde esophagoscopic passage of a filiform bougie, attaching a string and pulling it down through a stricture. Retrograde dilatation with the author's bougie was used, and at the end of eight months an 8 m.m. esophagoscope was passed through the mouth into the stomach and practically no evidence of stricture remained. The man was cured at the end of a year and the gastrostomy tube removed, the wound closing spontaneously.

are not as safe as esophagoscopic bouginage or continuous string-guided retrograde bouginage. Electrolysis has been used successfully by Guisez and dilatation with a heated bougie is advocated by L. W. Dean.

Retrograde Methods: The first successful retrograde catheterism was made by Erlich in 1897. Other names associated with retrograde methods are Bilroth, Abbe, Graham, Dunham, Guisez and Ochsner.

Retrograde esophagoscopy using the steel-stemmed filiform bougies of Jackson is of particular value in placing the string in gastrostomotized cases where it cannot be swallowed. Guisez used a single rubber tube, which was pulled up into the stricture from below. Ochsner used a doubled rubber tube, which was pulled on the stretch into the strictured area. The continuous string retrograde method utilizes the gastrostomy opening, and a continuous string through the esophagus, by means of which specially constructed, tapered, flexible, soft rubber bougies are pulled upward from below in increasing sizes.

In this brief discussion it would seem advisable to discuss only the methods which have given me the best results and which appear

Fig. 9. Child pulling up the retrograde bougie. Older children and adults frequently prefer to pull the bougie up themselves. This is allowed under the guidance of the physician.

to have the greatest element of safety in my own hands. First, peroral esophagoscopic bouginage of Jackson. This is especially of value in cases where there is a single stricture level, or where, if there are several levels, there is no serious difficulty in swallowing liquids. With local anesthesia in adults, and no anesthesia in children, the esophagoscope is passed down until the strictured area can be visualized. The steel-stemmed filiform bougies of Jackson are then passed by sight in increasing sizes, dilating the strictured lumen. Three sizes are used at each sitting, and the sizes are stepped up as the dilatation progresses. This method is ideal in cases where gastrostomy is not required.

Continuous String Retrograde Bouginage with the Author's Bougie: The author's retrograde bougie (see Fig. 5) is made in graduated sizes, from 10 to 34 Fr., running 10-12-14, etc., 13 sizes ir all. It is made 35 c.m. long and is made of the best quality of rubber molded over a continuous braided string. The braiding permits slight stretching of the rubber. When the bougie, on the stretch, is pulled into the stricture it becomes slightly smaller in diameter. When the pull is relaxed the bougie shortens and assumes its normal size. This outward force of the expanding bougie makes pressure directly outward against the flat surface of the stricture during the time the bougie remains in position. If the first size selected passes into the stricture easily, a second size may be pulled in. But if the first bougie fits tightly, only one should be used at each treatment. Treatments are given every three to seven days, depending on the progress made.

Following gastrostomy two or three weeks are allowed for the wound to heal, and during this period the patient is encouraged to swallow a string. In cases of complete aphagia under the rest given the esophagus, and proper feeding permitted by the gastrostomy the stricture will frequently open enough for the patient to swallow saliva and the string can be easily swallowed.

Stringing the Esophagus: When a patient in whom a severe stricture is shown by X-ray and esophagoscopy develops symptoms that indicate a gastrostomy will be required, it is best to get a string through the esophagus at once, even before gastrostomy, not for string-guided methods of dilatation, but to prevent complete atresia of the esophagus. The best method of placing a string is that of Mixter. A small size, No. 15 buttonhole twist, with a frayed end, is washed through with water. This will pass through a tight stricture when a knotted or shotted string will not pass. A bobbin of string is attached to the neck with adhesive after unwinding sufficient length to pass through the stricture. The end is passed through the anterior nares and nasopharynx and brought out of the mouth. The frayed end is then placed on the back of the tongue and washed down with water. When it is no longer regurgitated additional length is unwound from the bobbin and fed into the anterior nares, allowing the swallowed end to pass through the stomach. If the string gets into a snarl at any time and does not pass into the stomach it should be withdrawn and untangled or a new string substituted and then washed down again. After the string has passed into the stomach the length between the cardia and pylorus is easily withdrawn from the gastrostomy wound with a small right-angled re-

tractor (Jackson pillar retractor). After the string is recovered from the stomach, a larger size can be pulled upward through the esophagus and nasopharynx, the ends tied, and the string used as a continuous string. In cases that cannot swallow a string, retrograde esophagoscopy, using the steel-stemmed filiform bougies of Jackson to carry through a string, may be used. A ureteral catheter may be passed by retrograde esophagoscopy under fluoroscopic guidance and be recovered by peroral esophagoscopy. All methods of string placing should be tried early in severe cases before complete atresia occurs. After complete atresia has occurred the perforation of the obstructing tissue can be done under fluoroscopic guidance by peroral and retrograde esophagoscopy. The procedure has a very high mortality, however, probably 50 to 75 per cent, and every effort should be made to get the string through early before the esophagus closes completely. The length of time required for cure by retrograde dilatation varies from six months to two years. The patient requires hospitalization only, however, until the gastrostomy wound heals. It can then be treated as an out-patient. Gastrostomy feeding is used until the stricture is dilated up to 18 or 20 size dilator. Water is given by mouth at all times, however. After the esophagus is dilated sufficiently for the patient to swallow food that has been passed through a fine sieve, gastrostomy feeding can be discontinued, and the patient fed entirely by mouth. The gastrostomy tube should be worn, however, for a period of six months after the esophagus has been fully dilated. By taking this precaution, if the stricture has a tendency to recur and the esophagus becomes blocked with food at any time, gastrostomy feeding can be continued until the food can be removed through the esophagoscope. Esophagoscopic inspection is advisable after dilatation is complete before the gastrostomy tube is removed. Before the patient is discharged as well there should be practically no evidence of scar on esophagoscopic examination. The gastrostomy fistula usually closes spontaneously. If it does not, the closure is only a minor surgical procedure. We have seen no ill effects on the prolonged maintenance of the gastrostomic fistula. The method has been in use in our clinics eight years and a large number of cases have been treated with at least 90 per cent cured in uncomplicated cases and the other 10 per cent still being under treatment and progressing to cure.

The rationale of retrograde dilatation may be summarized as follows:

1. As noted first by Bilroth, the esophagus is dilated or bowl-shaped above and funnel-shaped below the stricture. This being the

case, we can enter the stricture more easily from below after passing the hiatus. The strictures are frequently multiple, and at different levels, with eccentric lumina. With the retrograde bougie, all strictures, regardless of number, are dilated simultaneously (see Fig. 4).

2. Patients, especially children, are more tolerant of retrograde than peroral instrumentation, and will allow the bougie to remain longer in position if pulled up from below than if brought down from the mouth (see Fig. 5).

3. The removal of the bougie upward when the full size is engaged in the entire strictured area requires less pull on the string than downward removal, due apparently to the reverse peristaltic action of the esophageal musculature excited by the movement of the bougie in the esophagus.

4. This method is carried out without anesthesia.

5. Hospitalization is not required for the carrying out of bouginage after the gastrostomy wound is healed.

6. Retrograde bouginage with the author's continuous string method gives the maximum of safety, because the danger of rupture is avoided if proper care is used in the amount of force exerted in the pull on the string, and if the bougie sizes are not "stepped up" too rapidly.

Complications: No complication has resulted from retrograde dilatation as far as I know. A bougie was lost in the stomach, due to the breaking of a string, but this would have been avoided if a string had been attached to the loop on the lower end of the bougie.

CONCLUSIONS.

1. The final diagnosis in "stricture" of the esophagus should be made by Roentgen ray study and direct esophagoscopic inspection.

2. The truth expressed in Trousseau's observation that "sooner or later all cases of esophageal stricture die of the bougie" is a clear statement of the dangers attending the blind pushing downward of peroral bougies. Blind bouginage should have no place in either the diagnosis or treatment of stricture of the esophagus.

3. Esophagoscopic bouginage of cicatricial stenosis is safe in careful hands and should be used in every case where gastrostomy is not required for the difficulty in swallowing. Esophagoscopic dilatation by sight is especially valuable in the treatment of stricture complicating an ulcerative lesion of the esophagus, as in peptic ulcer, tuberculosis or blastomycosis, etc. Local medication can be applied to the ulceration at the same time the dilatation is made.

4. Rapid instrumental dilatation of cicatricial stenosis of the esophagus is dangerous.

5. It requires no argument to demonstrate that drawing a bougie through a strictured esophagus by a string which the bougie must follow is incomparably safer than pushing any bougie could possibly be. The only possible danger would arise from the mistake of violently pulling through an oversized bougie.

6. It is true that retrograde treatment requires a gastrostomy, but this is a relatively minor operation that can be done quickly and, if necessary, under local anesthesia. Moreover, the giving of all food by mouth being stopped, the esophagus is relieved from the static esophagitis which is so often a factor in increasing the amount of cicatricial tissue, the subsequent contraction of which renders the stricture more and more difficult to cure.

7. The gastrostomy tube should be worn for at least six months after dilatation of the stricture is complete. The esophagus should be inspected by passing an esophagoscope before the gastrostomy wound is allowed to close.

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USES OF STRYPHNON GAUZE IN RHINOLARYNGOLOGY AND AURAL SURGERY.*

Dr. L. Forschner, Vienna, Austria.

In 1922 P. Albrecht, of Vienna, published the results of his experience in arresting capillary and parenchymatous hemorrhage with the aid of a preparation which is chemically the final product prior to synthetically prepared adrenalin. Owing to its vasoconstrictive action he assigned the name "Stryphnon" to the remedy, and he employed it at first in the form of a solution administered *per os* or as an injection. It first came into general use in the form of the so-called stryphnon gauze. Of considerable importance was the proof of the absolute harmlessness of the new preparation, which Bartel had been able to establish by microscopic investigations of a part of the liver which had been tampooned for a longer period with stryphnon gauze. P. Mayer published, in 1930, the results of investigations made in the Sauerbruch Clinic, in which he claimed to have demonstrated microscopically injuries to tissues which had resulted from stryphnon gauze being left in contact with them for some time; later investigations by Lerch and Mandl, however, showed the harmlessness of this agent; likewise, Sicher contested its alleged injurious action on the tissues.

According to Albrecht's investigations the activity of stryphnon resembles that of adrenalin by its vasoconstrictive action, but its effect becomes exhausted before the tissues have become impaired by the anemiation; on the other hand, the effect is more lasting than with adrenalin, so that small and smallest vessels can become thrombosed. Those first reports by Albrecht were very soon followed by a number of publications in a few special branches of surgery, and as regards the use of this preparation they were all couched in favorable terms. In otolaryngology Frey first reported on the use of stryphnon gauze and he recommended it for operations in that special branch. G. Hofer, Sperber and Weil confirmed these favorable observations, and thus the gauze became introduced into aural and rhinolaryngologic surgery. Shortly after these reports were published we began to use stryphnon gauze at the Ear Station of Wieden Hospital. Having employed it for seven years we are so satisfied by

*Report from the Wieden Hospital Ear Station.

now that we should scarcely care to be without it, so that also the publication of the results of our experience seems justified.

We use the gauze chiefly in the form of so-called stryphnon bandage contained in a slit carton as marketed, together with the gauze, in packets, by the Pharmaceutical Industry Co. of Vienna and Klosterneuburg. For rhinolaryngologic surgery and larger aural operations we use the selvedged gauze strips, 5 c.m. breadth, while for operations in and through the auditory canal (endotympanal) we use the strip 1 c.m. in width. In our surgical branch this size has proved more economic and is better adapted to sterile work than the large spreads of gauze of the packets, which mainly come into consideration for extensive operations. We cut small pieces 5 to 10 c.m. in length, the rest remaining sterile within the slit which the box contains. We also use stryphnon powder, as manufactured by the same firm, in certain operations of the nose and throat mentioned below.

We will now consider the particular operations where we specially prefer the use of stryphnon gauze to arrest hemorrhage. First, we have to consider all endonasal operations, *viz.*, submucous resection of the septum, Hirsch's perseptal opening of the sphenoid bone, West's lacrimal sac operation, the radical operation of the maxillary antrum, Halle's operations, Hajek's curettage of the ethmoid. All these operations necessitate the employment of a good styptic so as to render the field of operation free from blood and readily surveyable. The gauze under consideration meets these requirements in the highest degree. A disturbing feature when first using it is the grayish-yellow discoloration of the tissues which it brings about. However, the operator soon becomes accustomed to it and finds it no longer disturbing. The anemia produced is perfectly ideal and facilitates deep operations in a most remarkable manner. In all the hundreds of operations we have performed, we have never observed a case of injury from the preparation, or any injury of the tissues. No injurious effects ever did result when left lying for a considerable time. We use the gauze as a tamponade in profuse hemorrhage after operations on the turbinate bones, first insufflating iodoform powder into the nose. By these means it is possible to leave the tampon in the nose for one to two days without discomfort to the patient from unpleasant odor. One can also first insufflate stryphnon powder and afterwards plug with iodoform—a cotton plug in the front part of the nose is sufficient.

I should like to refer here to the good service which the gauze rendered in a case of most severe arterial after-hemorrhage following Hajek's ethmoidal operation. Such after-hemorrhages are known

to be extremely rare and are but scantily mentioned in literature. On that particular occasion it was necessary to retain a tampon in the nose during several days in the above described manner, without any untoward after-effects occurring. Precisely this case afforded special proof of the excellent value of the remdy. A second instance may be added, which was a case of severe hemorrhage from the drum membrane continuing for four days after a necessary paracentesis in a hemophilic young man. Also in this case plugging the auditory canal with the narrow stryphnon bandage (1 c.m. wide) rendered excellent service.

In every case of tonsillectomy we place in the site of operation, after excision, a larger strip of loosely-folded stryphnon gauze to which, on principle, we have more recently attached a long silk thread, and after a few seconds the wound, which in the meantime has become free of blood, can be inspected as regards a larger arterial hemorrhage, which is then arrested by surgical methods (acupression, ligation). The actual domain for the employment of stryphnon gauze are those threatening looking hemorrhages from the base of the tongue, which arise from dilated veins, to which O. Meyer has recently drawn attention. In such a case we suture an iodoform gauze tampon to the palatine arches and then tampon the space between the lingual tonsil and the sutured iodoform gauze by means of stryphnon gauze (armed with a silk thread); in this manner in conjunction with the vasoconstrictive gauze, a compression is exercised at the same time, and the hemorrhage is almost instantly arrested. In the parenchymatous after-hemorrhages due to loosening of the fibrinous wound membrane in one to six days after tonsillectomy, we have employed stryphnon powder on several occasions with marked success. In the rare cases of hemorrhage from the nasopharynx after ordinary adenotomy, and also in the more frequent cases of hemorrhage after excision of benign or malignant tumors by means of a forceps or adenotome (exploratory excisions), a firmly folded strip of stryphnon gauze is placed behind the uvula with the finger and pressed against the roof of the pharynx, where it is allowed to remain as a "lost" tampon for 24 hours or longer. Here also it is advisable to insufflate iodoform powder into the nose frequently.

In aural surgery we use the gauze in the form of narrow strips for tampons in hemorrhage after various operations in the auditory canal, the drum membrane, and the tympanic cavity, and also, especially after exploratory excision and extirpation of benign or malignant tumors and extraction of large granulations, in the middle ear

(polypi). The tampon, preceded also by an insufflation of iodoform powder, is allowed to remain there for 24 hours without injury. In the more extensive operations of aural surgery we use stryphnon gauze in a different manner. Here we take advantage of its anemiating character chiefly for the purpose of obtaining a good survey of the field of operation. It renders excellent service in mastoidectomy in cases of hyperemic bone, and when the bone is filled with granulations that readily bleed, as is especially the case in the so-called early operation for mastoiditis. We use the gauze in such a manner that a small stryphnon gauze tampon is left to press upon the bleeding region. In the meantime we operate at another spot, then rendering this spot in its turn free from blood in the same manner and returning to the first spot. When hemorrhage occurs in opening the antrum for the radical operation, we introduce a small piece of stryphnon gauze and leave it there for a few seconds, and after the field of operation has been rendered bloodless, we can then continue the work. We adopt a similar procedure in all cases of parenchymatous hemorrhage after opening up the tympanum and the apica. Here also the bone acquires a characteristic yellowish discoloration which the operator can readily put up with, if he appreciates the advantage of working with a good survey of the field of operation, which is achieved by employing the anemia producing gauze. In case the dura has been exposed, or a sinus is bare, we have refrained from the employment of stryphnon gauze, for although, as already stated, no injuries from its use ever occurred in our operations, yet we prefer to avoid any possible corrosive action on these very delicate structures. Likewise we have stuck to the use of iodoform gauze in the sinus operation proper, and we never use stryphnon for the tamponade of the mastoid process.

Summarizing, we may say:

1. Stryphnon in aural surgery and rhinolaryngology is most suitably used in the form of selvedged bandages of different widths, and also as powder to be insufflated upon bleeding tissues.

2. We have found stryphnon gauze extremely valuable as a styptic and for rendering the field of operation anemic and therefore providing the possibility of a better survey; also as a tamponade to stay in position for some time in combination with iodoform powder. We desist from using it as a tamponade for longer duration in the neighborhood of sensitive parts, such as the brain and sinus only, as in view of the investigations already referred to, the possibility of a deeper corrosive action must not be excluded. We have *never observed any injury* resulting from its use in the manner here described.

Its sole disadvantage is the characteristic discoloration of the tissues, to which, however, the operator soon becomes accustomed, being no longer a disturbing feature.

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International Digest of Current Otolaryngology.

Woloschin, in the Zeit. f. H., N. u. O., 28:68:1931, discusses the topography and anatomy of the mastoid emissary vein. The vein traverses the bone between the mastoid cells; it rarely goes through cells. In the event of its going through a cell, it usually occupies and fills the cell. The vein runs from outwards in, from upwards down and somewhat laterally in relationship to the sagittal bone. It runs in its bony canal, joins up with the diploic veins to the number of about one to three; the length varies between 0.5 and 2.24 c.m. on the right side and 0.63 to 2.93 on the left side. It is frequently in direct contact with the cells of the mastoid. The blood stream is directed from outwards in towards the sinuses and only rarely from the sinus outward going to the external jugular vein. Kelemen.

In the *American Jour. of Diseases of Children,* for March, 1931, Kaiser reports a study of the relation of tonsils and adenoids to infections in children. This was a control study of 4,400 children over a 10-year period. His conclusions were:

A. The removal of tonsils and adenoids influences favorably the incidence of the following infections: *1.* Cold in the head. *2.* Sore throat. *3.* Cervical adenitis. *4.* Otitis media. *5.* Rheumatic disease. *6.* Diphtheria. *7.* Scarlet fever. *8.* Nephritis. *9.* Dental infections.

B. The following infections were influenced favorably to a slight degree or not at all: *1.* Chorea. *2.* Measles. *3.* Laryngitis. *4.* Tuberculosis. *5.* Malnutrition.

C. The removal of tonsils influenced unfavorably the incidence of the following infections: *1.* Bronchitis. *2.* Pneumonia. *3.* Sinusitis.

Rosenberger.

Ed. Note: Kaiser has previously reported (see Jan., 1931, International Digest) a detailed study on some two thousand cases. His present report confirms his earlier conclusions and is based on additional material.

Wise and Sulzberger, of New York, in the Nov. 15, 1930, issue of the *Journal A. M. A.*, write on urticaria and hay fever due to trichophytin. They have reported a case of hypersensitivity due to trichophytin and they deem it possible that trichophytin products of pathogenic fungi may be the cause of some asthmas and hay fever as well as urticaria. In view of the widespread incidence of ringworm infections, particularly those of the feet, it may be quite reasonable to consider the possibility of an ever present allergen in the investigation of these patients. They give detailed reports of a case of uncommon skin irritations, hay fever and occasional attacks of asthma. An intradermal test with trichophytin was performed and immediately a marked local and general reaction was obtained. As a result of this experiment they add trichophytin to the list of possible allergic manifestations that may be attributed to the presence of ringworm infections.

Liggett, of New York, in the May 9, 1931, issue of the *Jour. A. M. A.*, presents an interesting case of parasitic infection of the nose. The patient complained of marked nasal discharge, hawking of pus and some tickling sensation of the roof of the mouth. Vigorous coughing and clearing of the throat resulted in the expectoration of a living insect about 1 c.m. long. The appearance of insects occurred at about two to three-week intervals, only one or two insects appearing at one time. Examination showed a mucopurulent mass in the right choanal space, which proved to be an insect. Clinically the patient had a bilateral maxillary sinusitis. Irrigations were of no value. Radical operation was performed on both antra; left antrum essentially negative; right antrum filled with a mass of polyps, necrotic tissue and fluid pus. A careful but unfruitful search was made for larvae. Patient then complained of intense headaches, more apparent in the occipital region. The sphenoids were opened and the right side was found to contain mucopus, but no larvae. After washing the sphenoids, within 10 minutes the patient blew three live insects into the handkerchief. Washing of sphenoids twice a week was productive of larvae being blown from the nose. Patient was then treated by filling the sinuses with oil by displacement irrigation. This resulted in prompt recovery.

A specimen was sent to the Smithsonian Institute in Washington, and the Curator of Insects there reported that the specimens were larvae of the black carpet beetle. He added that they are exceptional, in that they take two years to reach adult stage. The insects were not reproducing in the patient and would have come out of their own accord in the course of time.

Tucker, of Philadelphia, in the May 9, 1931, issue of the *Jour. A. M. A.*, presents a case of chronic hoarseness (ulcerative laryngitis), showing tuberculosis and cancer in the same lesion. A very thorough and complete examination of the patient was made; Wassermann was negative, X-ray evidence of tuberculosis of lungs, and a small mass attached to the anterior end of the right cord in the larynx, which had the appearance of cancer. In spite of the evidence of tuberculosis a biopsy was decided upon and the histological examination of the tissue showed both tuberculosis and early cancer. The sputum was positive for tubercle bacillus, and because of the patient's general condition, a laryngofissure was not as yet attempted. Had the patient's general condition been better, the pulmonary tuberculosis should not have been a contraindication to the surgical removal of the cancer from the larynx.

In the Medico-Legal Section of the May 9, 1931, issue of the *Jour. A. M. A.*, an interesting case is brought to attention. It involves the loss of teeth during tonsillectomy. The operating physician removed two loose teeth after the patient was under anesthesia, but before proceeding with the tonsillectomy. The evidence brought out was that it was the usual and approved practice to insert the gag when, in the judgment of the physician, the anesthesia is deep enough to proceed, but yet not so deep as to cause rigidity of the jaws, which will prevent insertion of the gag. The patient will chew and bite on the gag and it is impossible to estimate before hand how much of this an individual patient will do. The physician-defendant claimed that the patient in this case chewed down on the gag and her teeth buckled; one tooth practically fell out on the lips and another was so loose that it was liable to come out and enter the patient's windpipe. The patient attempted to make a case against the physician by introducing evidence that the operating physician looked around after he put the gag in the patient's mouth. The defendant claimed that his looking around was part of his general responsibility, inasmuch as he was responsible for the general condition of the patient and must watch the patient's face and also the ether machine. Expert testimony supported the physician and the Supreme Court concluded that no inference of negligence could be drawn from the momentary glance of the defendant at the ether machine; to draw such an inference would be to hold that it was more important to protect the patient's teeth than to guard against possible death from over-inhalation of ether. The Supreme Court affirmed the trial court's verdict in favor of the defendant.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Regular Meeting, Nov. 14, 1930.

Nature of Stimulation at the Organ of Corti in the Light of Modern Physical Experimental Data (Author's Abstract). Mr. R. L. Wegel.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

Auditory Nerve Experiments in Animals and Their Relation to Hearing. Dr. Ernest G. Wever.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HARVEY FLETCHER: It is late so I will detain you but a moment. It all sounds too good to be true, and as both speakers were talking I had a good many questions to propound, but they have all been answered. I wish to congratulate the speakers for the presentations made, and feel that all of us are honored by having the privilege of listening to them.

There is one thing I should like to point out in connection with the last paper that probably would have been pointed out had there been more time. It is a theoretical confirmation of the experimental results Dr Wever has just reported on the question of why he did not get responses to the high frequency tones. If you follow to conclusion the mechanism which has been proposed, called the volley theory, you can show that for the high pitched tones the intensity of the volley current is very much less than for low pitched tones. The signal is preserved in the volley current in high strength for the low frequencies and is probably the principal thing upon which the mind depends for judging the pitch. When you get to high frequency, you may have a large number of nerve discharges but with only a slight variation of the intensity of bombardment, which is the same frequency of the signal, so that for such tones the place on the basilar membrane which is principally stimulated is probably the principal factor which the mind uses for determining the pitch. Thus the qualitative results obtained by Weaver and Bray were predicted before the experiments were performed. These results are in accordance with the theory of nerve conduction, which has been extinct for a long time.

That is all I have to say. I am very much pleased to have been here.

DR. C. W. BRAY: I wish to express my appreciation of Dr. Wegel's work. It seems to me that we have a great deal of evidence showing that the cochlea acts on the principle of resonance. However, I think we should keep open the question of whether or not particular nerve fibres carry particular pitches independently of the nature of stimulation.

I would also like to point out that the results of the experiments on the turtle furnish a further check against artefact in our work. It might be expected that the conditions which could produce errors would be the same in the turtle as in the mammals. Consequently, a difference in the experimental results in the two cases indicates that we are dealing with true nerve impulses and not with artefact.

DR. H. B. WILLIAMS: Both of these papers furnish opportunity for a great deal of discussion, but there are one or two points only that I would like to mention, and one or two suggestions that I might offer to Dr. Wever.

In the first place, there is no question whatever of the true physiological nature of the response that he has obtained. His experiments pretty definitely rule out the possibility of spread to the end organs. He might cocainize the auditory nerve, which would not in any way materially affect the electrical conductivity; but the experiments that he has done eliminate every probability that this is a physical artefact. The matter of possible mechanical disturbance producing variation in resistance might be eliminated by compensating the

current. It seems to me that what he has done is sufficient to convince almost anyone of the real nature of the fact. I would recommend that he get some alligators. They are easy to handle; their heads are a pretty good size, and he might find them a good cold-blooded type to work with.

As to the theoretical side, I do not see the difficulty of reconciliation of Helmholtz's idea with the facts described. Instead of thinking of the basilar membrane as a place where the analysis is performed, we would rather think of it as facilitating the transference of the nerve energy.

Mr. Wegel told of 1500 places along the basilar membrane as responding to definite ranges of frequency. If you divide that into the number of fibres of the auditory nerve—25,000, as given by Dr. Gould—you get about 16 or 17 nerve fibres for each of these 1500 places. The interesting thing is that in the human ear the upper limit perception of sound is 16,000 per second, and it would take about 16 fibres for the corresponding region of the membrane to enable you to hear that.

It has always been supposed that by performing this experiment as has been done, you would not get the auditory frequency through the auditory nerve. No one knows what the refractory period of this nerve is; but so far as its histology is concerned it is likely to be pretty much like all the others, and in all probability we will be driven to one of these theories; that is, to assume that in order to get this frequency up the auditory nerve it is necessary to think of the fibres as operating *en echelon*, each recovering while the others are performing their function.

I wish to thank the Section for the privilege of hearing these papers and participating in the discussion.

DR. E. P. FOWLER: There are two points I would like to call attention to, one in Mr. Wegel's paper and one in Dr. Wever's. First, if this "intensity discrimination" theory of Mr. Wegel's is correct, there should be a different intensity discrimination in the areas which are deafened, compared with those not deafened, but only if there is disease in the cochlear vibrating membrane (or its contents), and not if the source of deafness is non-nervous lesions. From my own observations, this is actually the fact, but I intend looking more carefully into the matter now that Mr. Wegel has so beautifully shown me the possible mechanism.

Second, has Dr. Wever tested the secretions of the cat's auditory nerve while pressure is applied thereto? And if so, what were the results, if any? It would seem that this method of attack might bring out some interesting data.

I have certainly enjoyed hearing these papers.

MR. WEGEL (closing): I had the privilege and the pleasure of seeing the experiment of my friends, Dr. Wever and Dr. Bray, performed at Princeton last spring and was immediately impressed with the validity of their observations. I was struck particularly with the high grade of inventive ingenuity which suggested such an experiment in the first place, and with the rigorous care in eliminating artefacts with which it was carried out. The results thus far obtained are extremely interesting and, contrary to an impression which has been created, has no bearing on the correctness or incorrectness of the theory of hearing which I have outlined. The outstanding observation is that all the frequencies of sound which are incident on the ear are transmitted along the eighth nerve. This might appear at first sight to suggest that all analyses of sound may be central, but it is to be noted that this kind of a mechanism of nerve conduction was postulated and worked out by Dr. Fletcher before the experiments of Dr. Wever and Dr. Bray were made. Clearly, therefore, though the experiments have never been done before, they do not as far as results to date are concerned in any way invalidate the Helmholtz theory of hearing.

Dr. Fowler mentioned the desirability of making measurements of pitch and intensity discrimination in the neighborhood of regions of lower acuity. Such experiments would undoubtedly be interesting if they could be done on an ear in which the affection which originally produced the gap was not in active process. This is usually attended by tinnitus, which makes the measurement rather difficult. I have attempted to make such measurements on my own ear

in the neighborhood of such a spot and found before I finished that I was actually making "masking" measurements on the tinnitus.

I wish to express my gratitude to the Section for the privilege of participating in these meetings. They never fail to give a much appreciated inspiration.

DR. E. G. WEVER (closing): I must say that I feel rather apologetic for taking so much time! I should have liked more discussion of these questions. Yet I felt that it was important to present to you the experiments in full so that you could judge of their validity. For, plainly, the results must be established firmly before any theoretical structure can be built upon them. That is the reason that I did not enter further into discussion of the volley theory. I am glad that Dr. Fletcher pointed out the ability of that theory to account for a falling off in the strength of the nerve response with increasing frequency. I did suggest that the nerve current is probably not constant in value over the frequency range, and my view is in agreement with Dr. Fletcher's discussion.

In regard to Dr. Williams' suggestion of putting the nerve out of action by the use of some chemical substance, I may say that we have tried novocain but without clear success. We think that the intensity of the response was somewhat reduced after several minutes, but the judgment by ear is not very accurate, and we have felt a little uncertain of the experiment. Novocain does not penetrate nerve tissue very rapidly, and one has to prolong the experiment for such a time that its significance is reduced. A checking experiment of this sort must be limited in time, lest other physiological changes enter to obscure the results. Hence we have not worked extensively along this line.

Finally, I wish to thank the Section for the privilege of being here this evening, and for the opportunity of hearing the very interesting and important paper of Mr. Wegel. I am in complete sympathy with the sort of analysis that he has made of the problem of cochlear response. Of course, as Dr. Bray has said, we must not regard the theory as final at all points, and must hold it subject to revision. But it is of great value to scientific progress to work along certain theoretical lines simply to find how far one may go with the data available at the moment, and in the present situation Mr. Wegel has shown that one may go a long way. I am happy to have had the privilege of hearing this paper.

DR. HUGH B. BLACKWELL: I wish to express the appreciation of the Section for the excellent papers read by Mr. Wegel and Dr. Wever. The experiment which Dr. Wever has described will rank as a classical one in otological research. On hearing these papers one cannot fail to be impressed by the modesty and sincerity of the authors. The intellectual honesty which they have shown should serve as an inspiration to all research workers.